# Three essays about health progress and economic development in Africa

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# Trois Essais sur les Progrès de la Santé et le Développement Économique en Afrique

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*Pour Papa.*

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# ABSTRACT

This dissertation aims at opening the black box of African health progress during and following the colonial period. It does so by making three contributions to the literature on health policies, health standards and fertility patterns, in the 20th century's Africa.

The first chapter compares the colonial strategies for health policies' provision and other colonial policies in former French West Africa between 1904 and 1958. Drawing on colonial archives and existing data, it gathers a unique dataset containing colonial inputs at the colonial district level: medical and educational staff, vaccinations, public work's expenses and conscription. There was a very general strategy as regards to the provision of colonial services. In this context, the allocation of health inputs was specific in two dimensions only. First, medical staff was used as a means of colonial "coverage". Second, there is a long-lasting effect of prevention leading to a "diversification" strategy for all health investments. Away from these specificities, the common factors to all investments have to do with the colonial administration's preference for path dependence, investments' returns to scale, the diseases' contagion risk and the demand for colonial services. This work also suggests that there was no specialization of districts in one type of investments.

The second chapter of this thesis looks at the relationship between adult height and under-five mortality in the context of the "double African Paradox" in West Africa. Africans are relatively tall in spite of extremely unfavorable income and disease environments. Moreover, their height stature decreased since the 1960's despite improving health conditions and a fall in under-five mortality rates. This study points to selective mortality, by bringing forward a positive correlation between mothers' height and mortality in the 1980's West Africa. It then estimates a new model of height differential between survivors and deceased. Results imply that selective mortality could be large enough to mask significant height increases in the 1980's West Africa. In high mortality contexts, anthropometric studies should discuss mortality levels and trends. More generally, results imply that the issue of selective mortality is crucial to assess the long-term impact of most health interventions.

The third chapter tackles another specificity of African health: fertility and gender preferences. It develops a new indicator of gender preferences based on birth spacing. Applying it to Africa provides evidence that son preference is strong and increasing in North Africa, whereas Sub-Saharan African countries display a preference for variety or no preference at all. Traditional family systems accurately predict the nature of gender preferences, while religion does not. Last, the magnitude of preferences is stronger for wealthier and more educated women.

*Cette thèse a pour ambition d'ouvrir la boîte noire que constituent les progrès de la santé en Afrique au XX$^{ème}$ siècle. Ce faisant, elle apporte trois contributions à la littérature portant sur les investissements de santé, les conditions de santé et la fécondité en Afrique au XX$^{ème}$ siècle.*

*Le premier chapitre compare la stratégie de l'administration coloniale en ce qui concerne les politiques de santé à sa stratégie pour d'autres politiques coloniales dans l'ex-Afrique Occidentale Française entre 1904 et 1958. Ce chapitre utilise des archives coloniales et des données existantes pour créer une base de données inédite sur les investissements au niveau des cercles coloniaux: personnels de santé et d'enseignement, vaccinations, dépenses de travaux publics et conscription. La provision de politiques coloniales était déterminée par une stratégie très générale et l'allocation des politiques de santé n'est spécifique que selon deux dimensions. Le personnel de santé était mobilisé pour "couvrir" le territoire conquis et l'effet de longue durée de la prévention menait à une stratégie de "diversification" pour tous les investissements de santé. En dehors de ces spécificités, les facteurs communs à tous les investissements coloniaux sont liés à la préférence de l'administration coloniale pour une dépendance au sentier des investissements, aux rendements d'échelle des investissements, au risque de transmission de maladies et à la demande pour les services coloniaux. Ce chapitre suggère aussi que l'administration ne visait pas une spécialisation des districts par type d'investissements.*

*Le second chapitre étudie la relation entre la taille à l'âge adulte et la mortalité avant cinq ans en Afrique de l'Ouest, dans le contexte du "double paradoxe Africain". Les Africains sont relativement grands, malgré un environnement sanitaire dégradé et des revenus relativement bas. De plus, leur taille à l'âge adulte a diminué depuis 1960, malgré une amélioration des conditions de santé, et une diminution de la mortalité avant cinq ans. Ce travail suggère qu'une partie de ce paradoxe s'explique par la mortalité sélective, en mettant en avant une corrélation positive entre la taille des mères et la mortalité, dans l'Afrique de l'Ouest des années 80. Ce chapitre propose une modélisation inédite du différentiel de taille entre survivants et décédés. L'estimation de ce modèle indique qu'on ne peut pas exclure qu'en l'absence de sélection par la mortalité, les tailles adultes auraient augmenté, plutôt que stagné, pendant les années 80. Dans des contextes de forte mortalité, les études anthropométriques doivent discuter des niveaux et des tendances de mortalité, afin de prendre en compte la mortalité sélective. Plus généralement, les résultats de ce travail impliquent que la question de la sélection par la mortalité est essentielle pour évaluer l'impact de long-terme de la plupart des politiques de santé.*

*Le troisième chapitre traite d'une autre spécificité de la santé en Afrique: le lien entre fécondité et préférences de genre. Ce chapitre développe un indicateur des préférences de genre fondé sur les intervalles de naissance observés. Cet indicateur est ensuite appliqué au cas de l'Afrique. La préférence pour les garçons est à la fois forte et croissante dans le temps en Afrique du Nord, alors que les pays d'Afrique sub-Saharienne sont caractérisés par une préférence pour la variété, ou par une absence de préférence de genre. Les systèmes familiaux traditionnels prédisent avec précision le type de préférence, ce qui n'est pas le cas de la religion déclarée. Enfin, la magnitude des préférences de genre est plus importante pour les femmes plus riches et/ou plus éduquées.*

**Keywords**: Africa, Colonial Policies, Diseases, Fertility, Gender preferences, Health, Height, Selection, Under-Five Mortality.
**JEL Classification**: I1, I3, I15, I18, J1, J13, J16, N37, O1, O55, O12.

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# INTRODUCTION

Developed countries have experienced an unprecedented improvement of their health conditions during the 19th and 20th centuries. One of the most obvious illustration of this progress is the decline in mortality observed during this period. Today, no consensus exists on the exact underlying causes of this improvement. Developing countries, and first and foremost African countries, are still lagging behind in terms of health standards. In 2006, the Africa Regional Director of the World Health Organization stated: "the vast majority of people living in Africa have yet to benefit from advances in medical research and public health. The result is an immense burden of death and disease that is devastating for African societies" (World Health Organization, 2006). Existing works provide insight into health progress that were made during the last decades in Africa. If huge progress were made, they were not enough for Africa to catch up: "countries in the Region have made progress over the past 10 years but most are still not on track to achieve the health and health-related Millennium Development Goals. The major challenges include scaling up the implementation of universal access, strengthening health systems, and effective action on the broader social and environmental determinants of health" (World Health Organization, 2014).

## Health and development

Health is a key question for economists. Indeed, "health is both human capital itself and an input to producing other forms of human capital" (Bleakley, 2010a). First, health is a measure of the well-being of populations. It is one of the essential dimensions of human development. According to the World Development Report (2000), poverty could be broadly defined "as encompassing not only material deprivation (measured by an appropriate concept of income or consumption) but also low achievements in education and health." As such, aiming at health improvements is justified on intrinsic grounds; better health is of value in its own right. Second, health and nutrition matter for productivity and economic growth. Indeed, they influence many other dimensions, such as education, productivity, fertility, which in turn also impact development. In this approach, the emphasis is put on the instrumental rather than the intrinsic

value of good health.

As a consequence, deficiencies in health and failures in health policies are recurrent hypotheses put forward to explain income differences across countries. According to Bloom and Canning (2000), "poor health is more than just a consequence of low income; it is also one of its fundamental causes". Gallup, Sachs, and Mellinger (1999) even argue that poor health conditions are the main determinants of under-development in African countries. According to them, health standards explain a substantial part of the difference between African growth rates and the average growth rates of other countries: "tropical regions are hindered in development relative to temperate regions, probably because of higher disease burdens and limitations on agricultural productivity".

At the micro level, the link between health and development exists both at child age and at adult age. Following Alderman, Behrman, and Hoddinott (2004), poor health conditions during childhood can affect schooling, through three main mechanisms. First, malnourished children may receive less schooling, in total. This could be the case either because their caregivers seek to invest less in their education, either because schools use physical strength as a proxy for school readiness, or because malnourished children may have higher rates of morbidity and thus greater rates of absenteeism at school. Second, by delaying the age of entry in school, bad health conditions may lead to lower expected lifetime earnings, through a later entry in the labor market. Glewwe and Jacoby (1993) show that undernutrition delays the age of entry at school, and negatively affects income at adult age. The third pathway from malnutrition to educational outcomes is the direct consequence of poor health on cognitive development. Miguel and Kremer (2004) find that "deworming" substantially increases health and school attendance in Kenya. At adult age, there is a direct link between health and income, through the saving of resources that otherwise would go towards dealing with diseases and other health issues. Moreover, individual health can have large consequences on the labor market. Health is indeed correlated with the labor supply, due to the modification of the dependency ratio, and with labor productivity, through illness. Schultz (2002) finds positive effects of height on hourly wages in recent national surveys from Ghana, Brazil and the United States. Moreover, the impact of health on income has been theorized in the "efficiency wage" theory (Yellen, 1984). According to this theory, nutrition and productive capacity are positively correlated. Last, poor health conditions also affect fertility preferences, following Becker and Lewis (1973)'s model for fertility decisions. Shorter life spans can change life cycle behavior, and reduce the incentives to invest in human and physical capital. This model predicts that, in case of negative health shocks, the quantity/quality trade-off leads to an increase in fertility, and a decrease in educational outcomes.

Compared with micro studies, which clearly establish that health and development are positively correlated at the individual level, macro studies are less conclusive. The identification in macroeconomic studies is very challenging. First, because third factors affect both health and productivity. Second, because income is likely to be a significant determinant of investments in health. Most useful contributions to the literature thus rely on large-scale public interventions as a possible source of exogenous variation. Bleakley (2010b) finds that cohorts born after malaria eradication in the U.S., Brazil, Colombia and Mexico had higher incomes as adults than the preceding generations, suggesting potentially large benefits of public interventions in developing countries where malaria is still epidemic today. Carstensen and Gundlach (2006) come to the same conclusion, showing that malaria prevalence has a substantial negative impact on income, in the sample of former colonies for which data on early settler mortality are available. On sub-Saharan Africa, Gallup and Sachs (2001) argue that wiping out malaria could increase the continent's per capita growth rate by as much as 2.6% a year. However, Brainerd and Siegler (2002) show that the 1919 influenza epidemic had a positive impact on per capita income growth across the states of the U.S., during the 1920's. Acemoglu and Johnson (2007) exploit major international health improvements to estimate the effect of life expectancy on economic performance. Drawing on the international epidemiological transition of the 1940's as a natural experiment, they use predicted mortality as an instrument for life expectancy. They find that the increase in life expectancy leads to substantial increases in population, but has much smaller effects on total GDP, and negative effects on GDP per capita.

Unsurprisingly, the African continent is missing from most of the previously detailed works. Even in the very general study of Acemoglu and Johnson (2007), Africa is not included in the base sample, "because of a lack of reliable data on life expectancy in 1940". Indeed, we are lacking precise knowledge regarding the long-term trends of health standards in Africa.

## Long-term health trends

### The determinants of long-term health improvements

In theory, two main channels can be identified to improve health and living conditions of a given population. First, public provision can improve the health environment, which consists both of infectious agents (prevalence of malaria for instance), and of the protection brought by health facilities and hygiene. The collective health environment is generally improved through the implementation of a *health system*, a set of resources, medical care (preventive and therapeutic), hygiene and sanitation. Second, health outcomes can be improved through an individual channel, i.e. nutrition and the private demand for health.

This dichotomy between public provision and private determinants of health can be found in Fogel's and Deaton's work. Both recognize that the two channels can play a role, but they

do not identify the same factor as the starting point of health development. According to Fogel (1994), the escape from hunger and premature death was mainly an escape from a nutritional trap where we could not work to produce food because we were too weak, and we were too weak because we could not work to produce food to make us strong. Nutrition was a key element of the "historical great escape", and led to an improvement in health standards. This process has been observed over the last three centuries in Europe and in the United States, with both increasing heights and a secular decline in mortality rates. This thesis, putting forward nutrition as the main channel for health improvements, clearly insists on the importance of income. Yet, private determinants are not the only channel linking living conditions and health. Even though Deaton (2007) acknowledges that private income affects health outcomes through nutrition, he surely insists on the importance of the immediate health environment. According to him, "public health measures, particularly the provision of clean water and better sanitation, were the fundamental forces for mortality reduction during the century from 1850 to 1950".

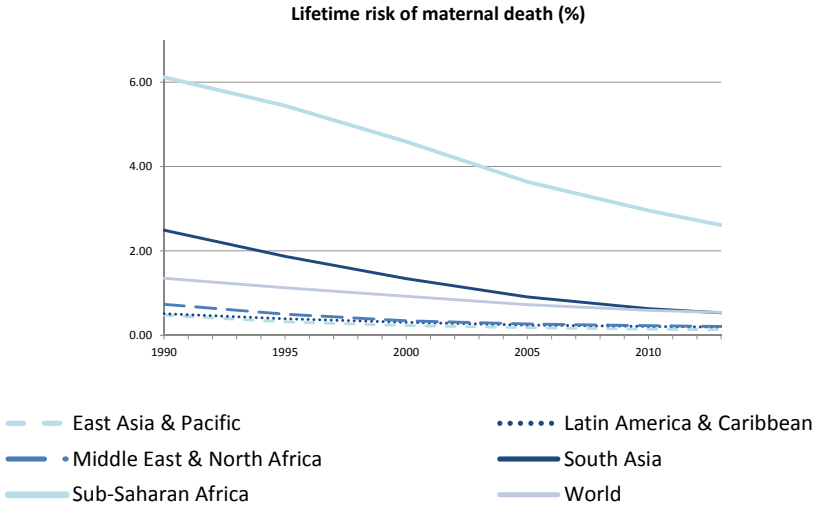
Depending on which channel is the most important between supply-side policies and private determinants of health, policy recommendations should not be the same. Of course, health supply and private demand are complementary. Whatever the level of demand, this demand needs a minimum provision of health to be met. Yet, if private determinants are the main force behind health improvements, the most efficient policy would focus on individual preferences and economic growth. On the contrary, if infectious diseases are the main driver of morbidity and mortality, public health policies should be targeted first.

**The evolution of health standards in Africa**

Describing what happened in Africa during the 20th century would contribute to shed light on what needs to be further done to improve health policies in the continent. Getting new insights on this question is particularly important in the context of recent health progress made in Africa. We know that Africa has the lowest level of health investments and the worst health conditions in the world today. What happened over the last decades, which could explain such a fact?

A first explanation is that the continent had extremely low health standards to start with, compared to the rest of the world. As a consequence, despite huge progress in health outcomes since colonial independence, it did not catch up with other regions of the world. The most salient example is the case of maternal mortality (Figure 0.1). Sub-Saharan Africa made huge progress from 1990. But it started from such high levels of risk that it was unable to catch up with other regions. The lifetime risk of maternal death was above six percent in 1990 in sub-Saharan Africa, followed by South Asia, with less than a 2.5% risk. The risk in sub-Saharan Africa is still 2.6% today, compared to 0.5% in South Asia. The same conclusion can be made

**Figure 0.1:** Maternal mortality in developing countries



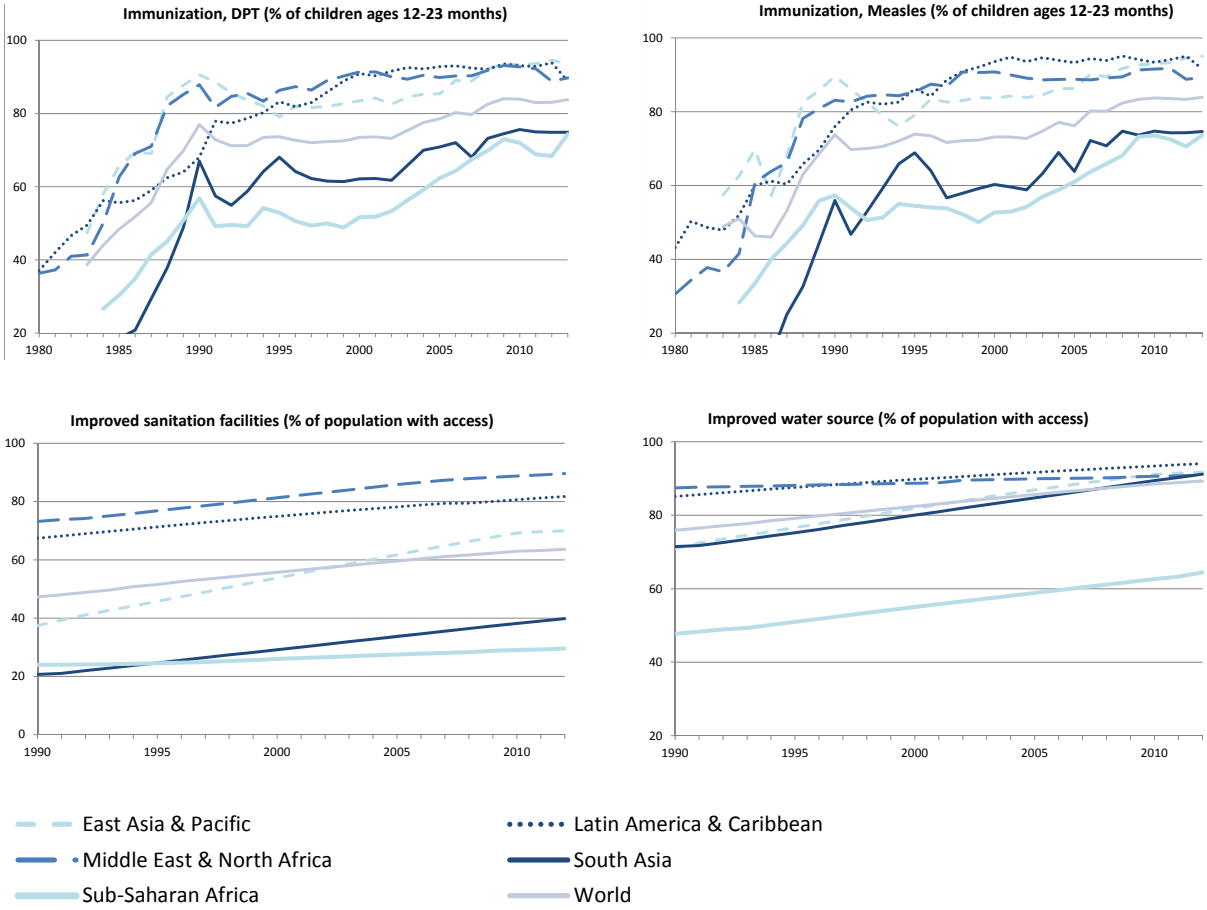

Author's own computation based on World Bank Data.

about DPT (diphtheria, pertussis, and tetanus), or measles immunization rates (upper graphs in Figure 0.2). South Asia and sub-Saharan Africa experienced the same trends as other regions over the last 30 years. However, both regions started from much lower levels, and are still lagging behind. Similarly, in sub-Saharan Africa, the proportion of population with access to improved sanitation, or improved water source, did increase as much as in other regions from 1990 (lower graphs in Figure 0.2). Yet, today, only 65% of the sub-Saharan population has access to an improved water source (30% has access to improved sanitation facilities), compared to around 90% in South Asia (40% for sanitation), its closest region.

A second explanation for Africa's low levels of health is that the continent experienced lower health progress than other regions of the world, for some health dimensions. These lower health progress could be related to the fact that welfare levels have been deteriorating in Africa. Chen and Ravallion (2004) estimate that the world poverty rate nearly halved between 1981 and 2001, declining from 40% to 21%. Meanwhile, the level of poverty in sub-Saharan Africa increased from 41.6% to 46.4% during the same period. While the under-five mortality rate decreased from 23% to 2.4% in Middle-East and North Africa from 1960 to 2013, the sub-Saharan mortality rate only decreased from 26% to 9.2% (upper graph in Figure 0.3). Similarly, sub-Saharan Africa and South Asia started from very close life spans in 1960: 40 years old in sub-Saharan Africa, and 42 years old in South Asia (lower graph in Figure 0.3). However, from the 1980's, the rate of increase slowed sharply in Africa compared to other regions of the world. Today, life expectancy has reached 67 years old in South Asia, an increase of 57%, compared to only 56 years old in Africa, an increase of 40%.

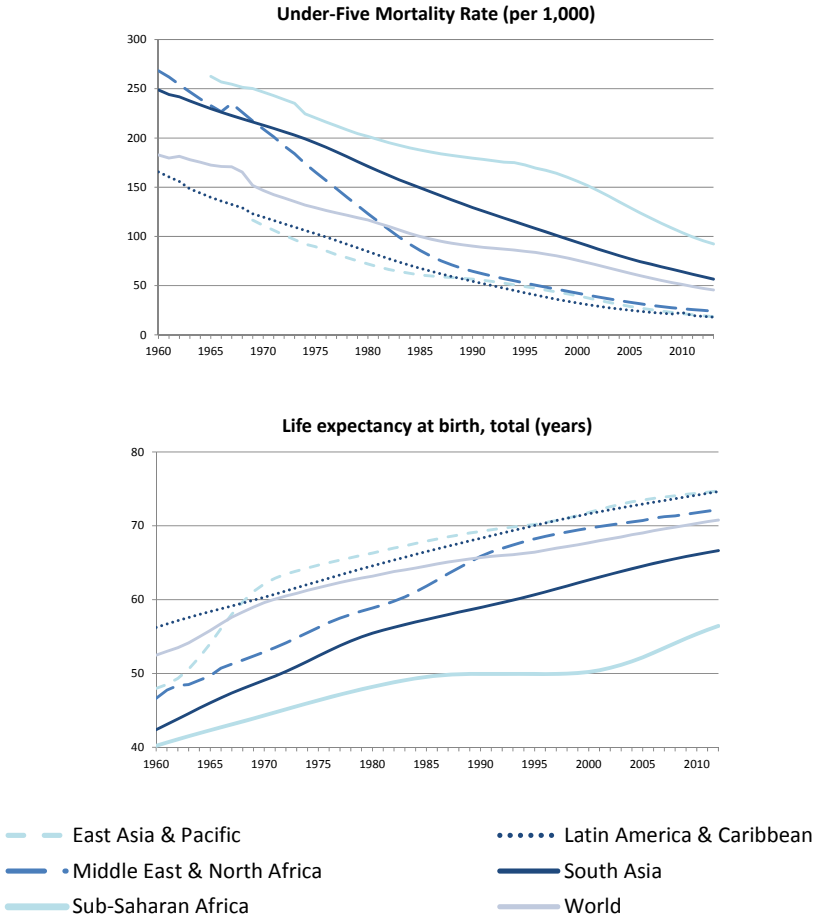
A third explanation is that Africa is still lagging behind in terms of health inputs. African countries represent twelve percent of the world population, but account for only one percent

**Figure 0.2:** Immunization, water and sanitation in developing countries



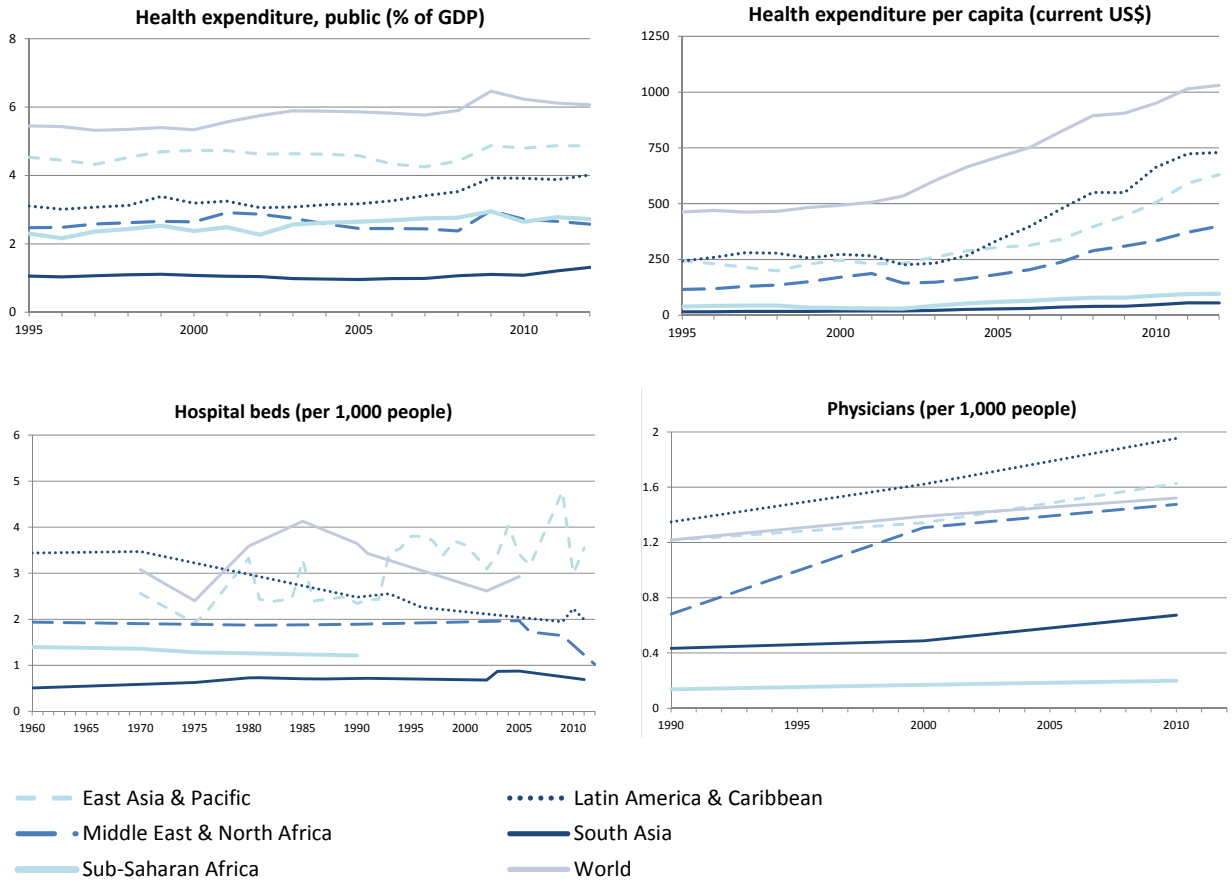

Author's own computation based on World Bank Data.

**Figure 0.3:** Under-five mortality and life expectancy in developing countries




Author's own computation based on World Bank Data.

**Figure 0.4:** Health expenditures and health inputs in developing countries




Author's own computation based on World Bank Data.

of worldwide health expenses and two percent of medical staff. There were no significant trends in health expenditures and health inputs over the last decades (Figure 0.4). Sub-Saharan Africa and Middle-East and North Africa approximately have the same level of public health investments as a percentage of GDP (upper-left graph in Figure 0.4), well above South Asia. However, when looking at the total health expenditure per capita, the picture changes, due to the relative disadvantage of sub-Saharan Africa in terms of GDP. Sub-Saharan Africa has the lowest level of total health expenditure per capita, together with South Asia (upper-right graph in Figure 0.4). The region also has the lowest number of physicians per capita in the world, and is second regarding the number of hospital beds per capita, after South Asia (lower graphs in Figure 0.4). Furthermore, the number of physicians per capita did not increase since 1990, while it did in all other regions.

These descriptive statistics give a view on health standards and their progress from the independence of African countries. But what happened before that? How did health inputs and outcomes evolve during colonial time? What can be said about the evolution of health standards along the 20th century?

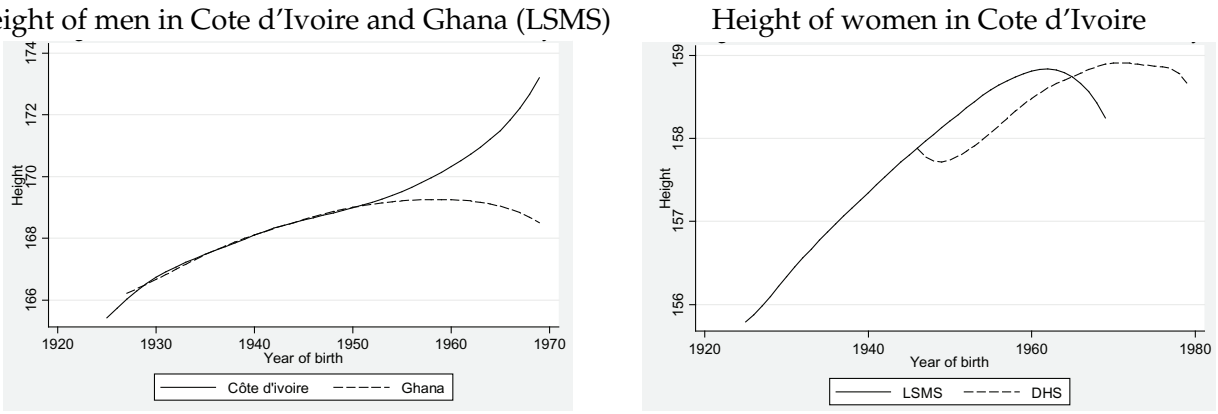
The study of the long-term evolution of health standards in African countries is very challenging because of a lack of reliable census data, even for the contemporaneous period. Very few births and deaths are officially registered in the continent. In particular, there are almost no mortality data for the first half of the 20th century. Until 1920-1930, only the European mortality is registered. From then on, the African mortality is sometimes reported in colonial data, but only when it happens within a medical institution, or when it is recorded by a member of the medical staff. Similarly, child mortality and birth rates are only available for a sub-sample of the population, which is not representative of the whole population.

Given this lack of data, standard measures of health – such as life spans or mortality – are not available to study the evolution of health standards in Africa; alternative measures need to be used. A few economic papers use anthropometric data to look at health standards in colonial Africa. For instance, Austin, Baten, and Moradi (2007) and Moradi (2009) on Ghana and Kenya and Cogneau and Rouanet (2011) on Cote d'Ivoire and Ghana, study long-term height trends. On former French West Africa, Huillery (2009) relates contemporary child z-score to early colonial investments in health, measured by medical staff. These works focus on one specific health outcome, height. However, as stated by Bleakley (2010a), "both theory and evidence suggest that we should stop thinking of health as a univariate object". Ideally, the study of the long-term evolution of health conditions should look at various dimensions of health: health policies on the one hand, and various health outcomes, on the other hand.

### This dissertation

The starting point of this dissertation is a research article, co-written with Denis Cogneau before I started this PhD. In Cogneau and Rouanet (2011), we show with survey data that the pace of increase in height stature experienced by successive cohorts born in Cote d'Ivoire and Ghana during the late colonial period (1925-1960) is almost as high as the pace observed in France and Great Britain during the period 1875 to 1975, even when correcting for the bias arising from old-age shrinking. By contrast, the early post-colonial period (1960-1985) is characterized by stagnation or even reversion in Cote d'Ivoire and Ghana. Trends are shown in Figure 0.5. This article argues that the selection effects linked for instance to measuring the height of women rather than men, mothers rather than women, and, most importantly, the interactions between height and mortality, cannot account for these figures. This paper provides evidence that a significant share of the increase in height stature may be related to the early stages of urbanization and cocoa production.

**Figure 0.5:** Height trends in Cote d'Ivoire and Ghana, 1925-1970




Building on this work, two research questions emerged. First, what is the magnitude of the selective mortality bias on adult heights? To what extent does the validity of anthropometric studies, and of some health interventions' impact evaluations, can be threatened by such a bias? Second, what can be said about health in Africa before 1960? How do health progress and health policies relate to the colonial presence? More generally, this dissertation aims at opening the black box of what happened to health in Africa during and following the colonial period. There is an urgent need to have a clear view on what happened in this continent regarding health inputs, health outcomes and fertility patterns, during the 20th century. This thesis tries to partly fill in this gap by investigating several aspects of the evolution of health conditions in Africa, making three contributions to the literature.

The identification of a causal impact of health interventions on various outcomes (productivity, income, health outcomes) is challenging. The first two chapters of this dissertation study two specific challenges in the African context. The first chapter addresses the issue of the endogeneity of health policies, asking whether the provision of health policies is specific as regards to other colonial policies. The colonial strategy for health policies' provision is compared with the strategy for other colonial policies, with a linear model and a two-tier model at the colonial district level. The second chapter tackles the question of selective mortality in the context of the "double African Paradox" in West Africa. It develops and estimates a new model of height differential between survivors and deceased, in order to assess the magnitude that could be reached by the selective mortality bias. The third chapter deals with another specificity of African health: fertility and gender preferences. It develops a new indicator of gender preferences based on birth spacing, which is applied to Africa. This chapter also studies the role of structural patterns and of socioeconomic factors in shaping gender preferences.

# CHAPTER 1

## THE PROVISION OF COLONIAL POLICIES IN FORMER FRENCH WEST AFRICA: ARE HEALTH POLICIES SPECIFIC?

**Abstract:**

This paper compares the colonial strategies for health policies' provision and other colonial policies in former French West Africa between 1904 and 1958. Drawing on colonial archives and existing data, it gathers a unique dataset containing colonial inputs at the colonial district level: medical and educational staff, vaccinations, public work's expenses and conscription. The allocation of such colonial expenses, studied in this paper, was neither random nor perfectly even across districts and underlines a very general strategy as regards to the provision of colonial services. In this context, the allocation of health inputs was specific in two dimensions only. First, medical staff was used as a means of colonial "coverage". Second, there is a long-lasting effect of prevention leading to a "diversification" strategy for all health investments. Away from these specificities, the common factors to all investments have to do with the colonial administration's preference for path dependence, investments' returns to scale, the diseases' contagion risk and the demand for colonial services. This paper also suggests that colonial investments' decisions were guided by a general – rather than specific – principle of investments' complementarity; and that there was no specialization of districts in one type of investments.

## 1.1 Introduction

Africa has experienced great health progress over the last decades. From 1950-1954 to 2005-2010, in sub-Saharan Africa, average infant mortality rates decreased from 18% to 8%, and life expectancy rose from 36.2 to 52.9 years.[1] Despite such progress, Africa still has very poor health inputs and outcomes. African countries represent twelve percent of the world population, but account for only one percent of worldwide health expenses and two percents of health agents. The African continent has very low levels of government health expenditure, which are compensated by a high rate of external resources (see Figure A-1.2, in the Appendix). These low levels can be partly explained by the fact that there were no significant trends in terms of expenditures and health inputs over the last decades. Figure 1.1 shows that sub-Saharan Africa made no progress regarding public and total health expenditures over the last 20 years. Similarly, there was no significant increase in the number of hospital beds and physicians per capita.

Today, African countries also have the worst mortality and morbidity indexes in the world. More than half of maternal mortality cases occur in sub-Saharan Africa, where the lifetime risk of maternal mortality is one out of $38$, compared to one out of $3,400$ in high income countries. Under-five mortality rates are of 10% on average in sub-Saharan Africa compared to 6% in South Asia (see Figure 1.2).

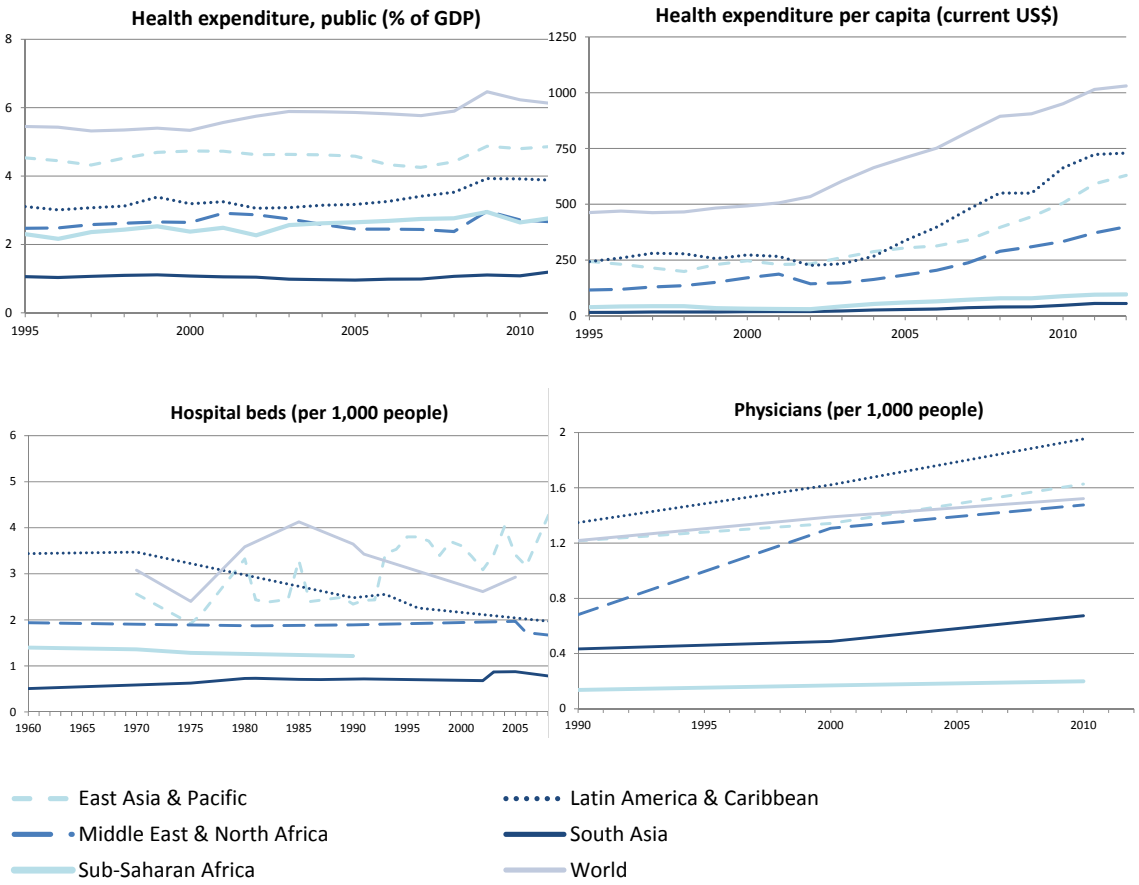
Africa faces very low contemporary levels of health standards in spite of relatively large progress over the second half of the 20th century. This observation leads to asking what happened to health standards in Africa before the 1950's.

The motivation to study African health before the mid-20th century also relies on the fact that there is no quantitative evidence on colonial health policies so far. French colonization lasted for more than half of the 20th century in former French West Africa. One of the ex-post arguments brought forward by those in favor of colonization was that it contributed to increase the population's health standards. More generally, Lasker (1977) states that "the Natives' gratitude toward the doctor creates for France unlimited rights to the land of Africa. The soldier pacified the country; the administrator, the engineer, and the planter made it live. The doctor, however, does more than heal the body, he conquers the hearts". If colonial health policies did "conquer the hearts", there exists no empirical evidence on their actual efficiency and on their way of operation. There is no relevant counterfactual that would allow to study the impact of colonization on health standards in West Africa.

Consequently, rather than assessing the efficiency of colonial policies, this paper takes a

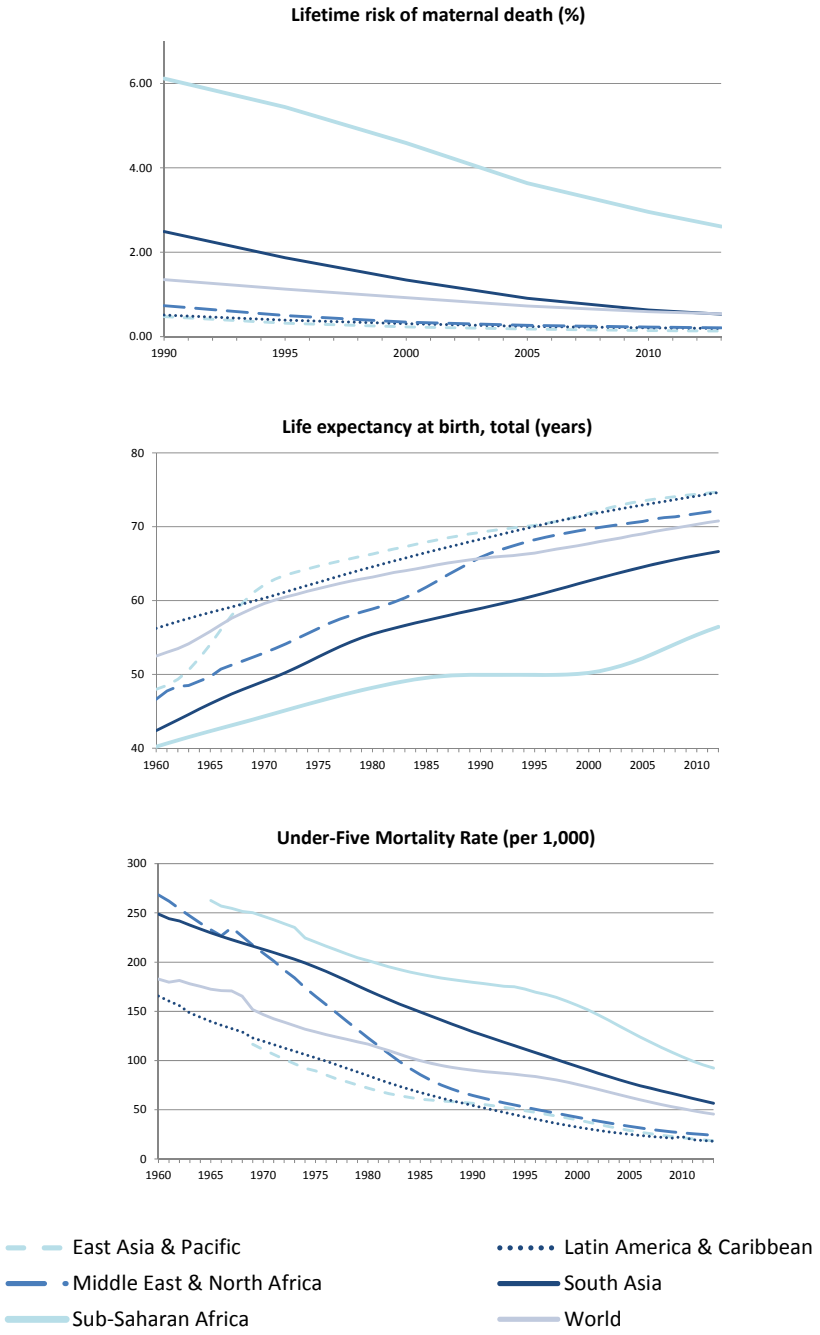
[1] According to the 2012 revision of UN World Population Prospects data.

**Figure 1.1:** Health inputs in developing countries




Author's own computation based on World Bank Data.

**Figure 1.2:** Maternal mortality and life expectancy in developing countries




Author's own computation based on World Bank Data.

different approach: it contributes to the literature by opening the black box of colonial health in sub-Saharan Africa. It does so by providing the first detailed quantitative analysis of colonial health policies and their determinants. These are compared to other types of colonial investments, namely education, public works and the military. The main research questions are the following: what kind of health policies did the colonial administration implement? On which aspects of health were the main efforts done? What was the main strategy for the provision of health? To what extent was there a specific strategy for health policies, as compared to other colonial policies?

The focus of this paper is former French West Africa. Detailed district-level data from the colonial health administration are gathered for Cote d'Ivoire, Dahomey (Benin), French Sudan (Mali), Guinea, Upper-Volta (Burkina Faso), Mauritania, Niger and Senegal, for years 1904 to 1958. These data contain detailed information on health inputs (staff and vaccinations) which allow one to follow the development of the colonial health system over time. They are matched to other colonial expenses' data, namely teachers, public works' expenses and the number of conscripts.

This paper describes the decision-making process of health provision. The panel of districts and their health inputs are considered as a health system, ruled by the colonial administration, and serving some of its interests. "The health system was not, or was much more than, a humanitarian effort; it served specific goals of the French rulers" (Lasker (1977), on Cote d'Ivoire). The allocation of colonial policies was neither random nor perfectly even across districts. This paper asks what were the main drivers underlying the allocation of colonial investments. More precisely, this work answers to three big questions regarding colonial policies' strategy. First, it looks at the impact of the existing colonial system on colonial investments' decisions. Are colonial investments always made in the same districts, or to the contrary aiming at covering the territory? Second, to what extent do investments target specific populations? Third, how do colonial investments relate to disease prevalence? Does disease prevalence capture health needs and contagion risk only, or the demand or "sympathy" towards colonial services as well?

The two-tier model of Cragg (1971) is used to disentangle between investments' extensive margin – the decision to have a positive number of inputs in a district – and investments' intensive margin – the decision on the quantity of inputs in the district. Empirical results show that there is a very general strategy as regards to the provision of colonial services. The common factors to all investments have to do with the colonial administration's preference for path dependence, investments' returns to scale, the diseases' contagion risk and the demand or "sympathy" towards colonial services. Moreover, colonial investments' decisions are guided by a general – rather than specific – principle of investments' complementarity. The allocation

of health inputs is specific in only two dimensions. First, medical staff is used as a means of colonial "coverage", to reach out to isolated territories. Second, the long-lasting effect of prevention leads to "diversification" for health investments: districts who got more health inputs in one period tend to have less in the following period. On the contrary, the allocation of teachers is self-reinforcing; it only relates to the health system through path dependence and returns to scale.

This article is organized as follows. The next section reviews the literature and describes the historical context of this work. Data are detailed in Section 1.3. Section 1.4 provides descriptive evidence on the spatial and temporal allocation of health provision. Section 1.5 presents theoretical scenarii for the determinants of colonial policies, as well as the empirical strategy. While the following section contains estimation results, the last section concludes.

## 1.2 Literature and historical context

The quantitative evidence on health conditions in French West Africa during the first half of the 20th century is very limited. Most qualitative works by historians or sociologists divide the colonial era in two periods regarding health standards (Domergue, 1981; Echenberg, 2002; Feierman, 1985; Lasker, 1977; Manning, 1998).

Existing qualitative works insist that the first period, going from 1890 to the 1920's, was characterized by epidemics and no population growth.[2] The consensus is that Africa underwent a population decline, or at least a population stagnation during the early colonial period.[3] According to the existing evidence, West African countries were doing slightly better among African countries: their population stagnated, or slightly grew. The final disastrous event that marked the end of early colonial population decline, or stagnation, was the influenza pandemic of 1918-1919, which took between one-and-a-half and two million lives in sub-Saharan Africa, and an estimated five percent of the population of francophone sub-Saharan Africa. Possible explanations are put forward in historical and sociological works, but are not empirically tested. Feierman (1985) describes the three basic interpretations of early colonial population loss or stagnation given by historians.

[2]The history of health in colonial India was characterized by a similar timing. According to Visaria and Visaria (1982), there were two distinct periods of population change in British India. First, 1871-1920 with a population growth rate of 0.37% and many mortality crises (famines and epidemics). Second, 1921-1941 with a population growth rate of 1.22% and the disappearance of mortality crises.

[3] There is no precise quantitative evidence on this because of a lack of reliable census data. In particular, there are almost no mortality data for the first half of the 20th century. Until 1920-1930, only the European mortality is registered. Since then, the African mortality is sometimes reported in colonial data, but only when it happens within a medical institution, or when it is recorded by a member of the medical staff. Similarly, child mortality and birth rates are only available for a sub-sample of the population, which is not representative of the whole population.

The first interpretation is that population loss in the beginning of the colonial period could be seen as a mere continuity with the previous period: the epidemics of the early colonial period were in no way unusual. According to Curtin's work, Africa was "the white man's grave". First waves of Europeans going to Africa were very much affected by tropical diseases.

A second interpretation is that some consequences of colonization impacted health. Urbanization, the building of roads and railways, labor migration (for instance, of rural populations to work sites) and the movement of armies could all have increased the possibilities of transmitting communicable diseases. For instance, Dozon (1985) argues that yard openings, as well as the development of a crop agriculture, led to population movements which seemed to increase the development of trypanosomiasis epidemics. However, Domergue (1981) argues that it is very unlikely that the development of commercial roads could explain the increase in trypanosomiasis prevalence during colonial times, given that the colonization did not lead to a great opening of new roads.

A third and last interpretation is that conquest was a political event which deprived Africans of the capacity to control their own environment. The European conquerors forcibly instituted new patterns of settlement, labor, and land use and could thereby have destroyed the basis of survival. For instance, Manning (1998) states that "forced labor and forced cultivation interfered with the normal African work patterns, and cut nutritional levels to the point where health was impaired".

The last two interpretations are also mentioned by Echenberg (2002) concerning colonial Senegal. Major causes put forward by historians regarding West Africa never include direct transmission of diseases from Europeans. On that point, the history of colonial West Africa cannot compare to the history of other African countries, such as Uganda and Congo. It does not compare either to the colonization of the Americas.[4] West Africans, together with Europeans and Asians, had a natural resistance to smallpox, measles, typhoid, and other diseases endemic to the Old World. West Africans had been in contact with Europeans long before the end of the 19th century, through the intermediary of trading posts. These diseases were still deadly but not as deadly as they would have been in a population that lacked this natural resistance.

[4] Wachtel (1971) describes the "shock of white men" for pre-colombian societies, which had been isolated from the rest of the world for centuries. Works such as Cook (1998) also describe the biological mingling of the previously separated Old and New Worlds, which led to the disappearance of entire peoples in the Americas. "Amerindians had never before experienced the deadly Eurasian sicknesses brought by the foreigners in wave after wave; smallpox, measles, typhus, plague, influenza, malaria, yellow fever" (Cook, 1998).

The second part of the history of colonial health starts in the 1920's, when the colonial administration becomes aware of population stagnation and bad health standards. This was partly a result of World War 1, which brought the French to the realization of their dependence on these colonies for both raw materials and manpower for defense (Lasker, 1977). In 1918, the French West African medical school Jules Carde was created in Dakar. In 1921, a new colonial plan was launched that put forward health and hygiene, for the "enhancement" of territories. The major obstacle to economic growth, as outlined by the Minister of Colonies Albert Sarraut, was the lack of sufficient native manpower: "the growth of colonial production is most especially, a question of labor, of preserving the population and birth rate; to be accomplished by a major program of hygiene, medical assistance, and education" (1923). A new doctrine is announced: "the quantity and quality of indigenous races shall be developed" (Governor-General Carde, Dakar 1930 in *Instructions du Gouverneur général Carde, relatives à l'orientation et au développement des services de l'assistance médicale indigène*). From then on, the objective is to change the curative action of the Native Medical Assistance,[5] which was created as soon as 1905, into a preventive action. This decision aimed at preventing diseases and epidemics, and at increasing population growth. Some historians indeed argue that the second part of the colonization, from the 1920's onwards and particularly after World War 2, was characterized by health progress, mortality decline and population boom. The causes of the mortality decline are largely unknown. One possibility is famine reduction through improvements in transportation. A second possibility is that medical interventions have saved lives (sulpha drugs, antibiotics, and anti-malarials, immunization, etc.). Feierman (1985) also insists that a rise in fertility could have contributed to the population boom. The colonial economy created great labor demands on domestic groups, whether in places which supplied male migrant workers or where export crops were important. The only way to cope was to increase fertility by shortening abstinence periods.

Lasker (1977) even sets apart a third period, the 1950's, during which health services continued to expand to meet the growing needs of the economy. However, as political stability again became an important consideration (in Cote d'Ivoire at least), there are indications that a slackening occurred in some areas in response to political opposition.

The few economic papers looking at health standards in colonial Africa focus on anthropometric data. Moradi provides several studies of the evolution of health standards in British colonial Africa: Austin, Baten, and Moradi (2007) and Moradi (2009) on Ghana and Kenya. In Cogneau and Rouanet (2011), we study the increase in height stature experienced by successive

[5] *Assistance Médicale Indigène*: the branch of colonial health services, supposedly present in every districts.

cohorts born in Côte d'Ivoire and Ghana during the late colonial period. We provide evidence that a significant share of the increase in height stature may be related to the early stages of urbanization and cocoa production. Huillery (2009), which is closer to this paper, uses child z-score as her main health outcome. She correlates early colonial investments to contemporary outputs. Focusing on specific channels, she shows that districts which had more teachers during colonial times still have better educational outputs today. Regarding health, she finds that child z-scores today are correlated with colonial times' medical staff. Rather than looking at one health input, medical staff, and one health outcome, child z-score, this paper considers health as multi-dimensional. It makes use of several health inputs: medical staff, but with the distinction between physicians and nurses, African and European staff, etc. It also looks at smallpox vaccinations as another input. Furthermore, using a cross-section, Huillery's paper does not look precisely into what happened during the colonial period. On the contrary, this paper uses panel data, which allows to study health inputs, other colonial inputs, and health outcomes within district and within the colonial period.

This paper also relates to the existing literature on the determinants of public policies, and more specifically on health supply determinants. In its paper on the 1880-1930 rise in social spending, Lindert (1994) compares several potential factors which could have shaped the rise of this spending. These include income growth, demography, democracy, and religion.

In a more specific work, Cain and Rotella (2001) ask what explains the large increase in American cities' expenditures on sanitation in the first 30 years of the 20th century. They start from the dominant view of urban government budgetary decision-making, the "incrementalist approach". This approach implies that, in general, municipal expenditure patterns change slowly and do not respond to specific conditions or to public pressures. According to the authors, the only exceptions to this approach come from dramatic crises. They show that, in their context, urban mortality shocks did lead to increases in municipal sanitation expenditures. Unfortunately, they do not test other possible channels which could explain city-level expenditure patterns. As they state, "a full explanation will include demonstration effects from other cities, technological imperatives, and a wide range of political factors such as reform movements and machine politics".

In the context of this paper, the most relevant work in this literature is probably the study of Costa and Kahn (2003) on public health investments in the U.S., from 1910 to 1930. Their main finding is that "in the early 20th century, U.S. support for redistribution was higher in areas with more black and immigrants". A higher share of blacks in the population actually raised voters' demand for public sanitation and disease control. This is in contrast with several

empirical studies on more recent periods, which documented that support for redistribution was lower in areas where more minorities live (Alesina, Glaeser, and Sacerdote, 2001). The argument made by the authors to reconcile these findings is that before World War 2 the middle class was motivated by self-interest – saving white lives – to support large public health investments. After the war however, policymakers did not see a clear case for providing blacks with broader kinds of public health and income security, whose direct impact on white lives was considered as smaller. As discussed in Section 1.5.2, a similar self-interest, but on the part of Europeans rather than of the middle class, could have explained the allocation of health provision during colonial times.

## 1.3 Data

### 1.3.1 Colonial data

To describe health provision and identify its main drivers during the colonial period, this paper uses an original dataset extracted from annual reports of the colonial administration of former French West Africa. The *Série 2G*, in French colonial archives, is kept in Dakar. However, its microfilms are also kept in the *Archives Nationales* in France. These archives contain detailed health reports for each colony and each year between 1904 and 1958. Only data that were available at a more disaggregated level than the colony (district level) were gathered.[6]

These colonial annual health reports describe precisely health investments, as well as some health outcomes. The main information extracted from these reports concerns medical staff (Europeans and Africans, physicians, nurses, etc.), medical facilities (hospital beds), vaccinations per disease (smallpox, yellow fever, tuberculosis, plague, etc.), medical care (assisted deliveries, consultations, hospital admissions, etc.) and health outcomes (cases and deaths per disease).

During the colonial period, other potential health providers are:

1. Army services, administering vaccinations.
2. Catholic missions, which provide basic care.
3. NGOs, mainly from the 1950's.
4. Private practitioners, although according to the *Annuaire Statistique de l'AOF*,[7] they are very rare. There are only 19 private practitioners in 1946 in AOF and 65 in 1955.[8]
5. Traditional medical practices.

[6] In the Appendix, Figure A-1.1 shows an example of tables from which data were extracted.

[7] *Annuaire Statistique de l'AOF 1950-54*: Volume 5, Tome 1, which was Published by the Haut-Commissariat de l'AOF, Direction des Services de la Statistique Générale et de la Mécanographie.

[8] According to the *Annuaire Statistique de l'AOF* (op.cit.).

The colonial administration could also have had an impact on health through other kind of public policies than those described in the reports, such as sanitation programs or famine prevention. There are very few records of such policies in colonial health reports, apart from a few sanitation programs in major West African cities, Dakar in particular. Historians' works do not record any major policy of this kind either. in any case, colonial health policies described in these reports were reaching a much greater proportion of the population than any kind of sanitation program. This comes from the fact that access to colonial health care was free. In 1931 Senegal, less than 0.1% of the Native Medical Assistance's budget came from non-free health care.[9] The main incomes for health care are medical assistance taxes and hygiene taxes.

Consequently, one can make the plausible assumption that colonial health policies described in these reports form the vast majority of the investments that were made in modern health care and medicine in colonial French West Africa.

Health investments are compared to three other types of colonial expenses: investments in education that are proxied by the number of teachers, public works' expenses and the number of military conscripts. Teachers' and public works' data are the same data as in Huillery (2008). They were extracted from colonial archives. The number of conscripts is extracted from data gathered with Denis Cogneau and Alexander Moradi on military conscripts in former French West Africa (1880-1960). We built a sample of military conscripts files for all the region. In addition, we digitalized annual army reports which contain aggregated statistics at the district level. The number of enlisted soldiers per district and year is used as a proxy of military expenses; it is extracted from these annual army reports, when available. When not available, individual conscript files are used to compute an estimated number of enlisted soldiers.

This paper also makes use of data recently gathered by the e-Geopolis project about urbanization in Africa. These data provide estimates for the size of each country's main cities (with more than 5,000 inhabitants around 2000) for each decennial year between 1920 and 1970. For other years, urban population is interpolated, assuming exponential growth, except when the initial urban population is zero, in which case a linear interpolation is applied up to the next decennial year. The aggregation of these data at the district level provides a dataset of urban population for each district and period.

Ideally, in order to correctly assess the availability of colonial services, each individual should weigh the same in the sample. All regressions and aggregated statistics are thus weighed by the district population. District-level population data are the same data as in Huillery (2008). She provides data on the total population, as well as a breakdown accounting for Africans and

[9] From *Budget de l'hygiène publique et de l'assistance médicale indigène, Compte définitif des recettes et des dépenses*. General government of former French West Africa.

Europeans. Consequently, African (resp. European) specific variables are weighed by African (resp. European) population.[10]

### 1.3.2 Colonial statistics: main challenges

The previous section argued that colonial policies form the majority of Western-style health inputs in colonial French West Africa. However, what the colonial reports provide is the *record* of these policies, as reported by the colonial administration. Data extracted from these reports cannot therefore be considered as *given*. They were produced by the colonial administration, i.e. by the very same administration which was implementing health policies. This is a very general concern with administrative data, which also applies to other types of colonial investments and which has specific consequences in the context of this paper.

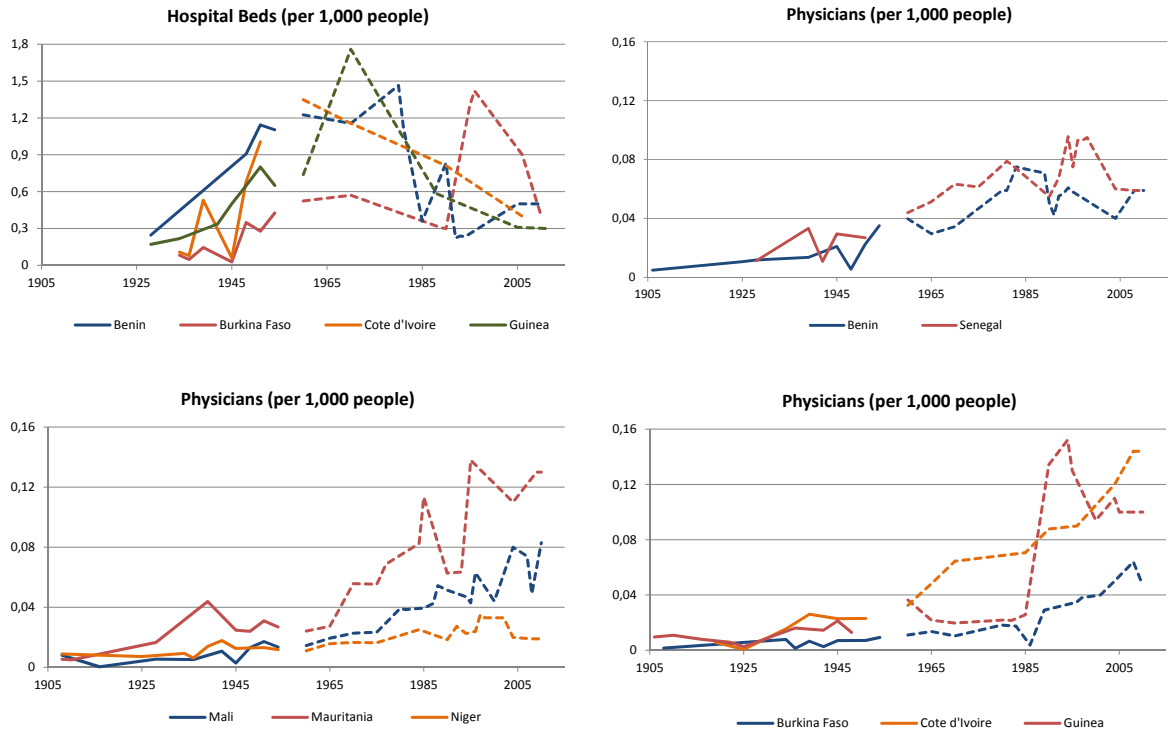
There are several matters regarding the very aim of colonial statistics that question their reliability. Colonial statistics, and first and foremost colonial health statistics, could have been used as a political tool. For instance, they could have been used as a propaganda in favor of colonialism, or as a governor's tool to show good outcomes at the local level. Vaccination campaigns during colonial rule were one of the earliest and most extensive public health programs that colonizers bragged about as evidence of the advantages of colonial rule. According to Chippaux (1980), smallpox vaccinations were described as "an appreciable agent of propaganda" by French colonial medical officers.

Apart from political incentives, one has to be careful in interpreting these health outcomes variables as there could be some measurement error biases due to reporting or to observation. In the *Annuaire Statistique de l'AOF* (op.cit.), it is reported that "a rise in the number of observed cases often comes from a deeper action of health services, and does not necessarily lead to the conclusion that a disease did spread out.". Hence, if more cases are being observed, this could be explained by better tracking. In the same *Annuaire Statistique*: "hospital medical care simply indicates the level of development of health services in the district", implying that health demand would not impact medical care statistics. On the contrary, Lapeyssonnie (1988) insists that an increase in consultation numbers would indicate a greater trust towards colonial medicine, or more generally towards European medicine.

All these concerns imply that colonial data used in this paper should be considered as a statistical construction from the colonial administration rather than the exact description of health inputs, health outcomes and other colonial inputs during the colonial period. The best way to test the reliability of colonial data is to compare them to other sources.

[10] These population data are interpolated assuming exponential growth, apart from European population, for which linear growth is a more plausible assumption.

**Figure 1.3:** Health provision per capita: colonial vs. World Bank data




Author's own computation: dashed lines are extracted from World Bank data, plain lines are extracted from colonial data used in this paper.

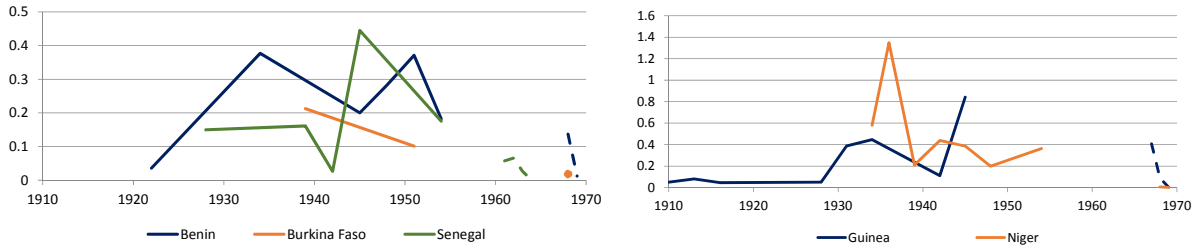
Health inputs' levels can be compared to country-level statistics provided by the World Bank's World Development Indicators database for the immediate post-colonial period. Figure 1.3 provides such a comparison. The upper-left graph shows the evolution of the number of hospital beds per capita in four former French West African countries. There is a clear continuity at independence for both statistical series, colonial statistics used in this paper (plain lines) and World Bank data (dashed lines). The same conclusion can be made for the number of physicians per capita, whose trends are shown in the three remaining graphs of Figure 1.3. These show that inputs' levels from both series are comparable around colonial independence. These findings are very reassuring regarding the quality and objectivity of colonial data. As the French colonial administration and the World Bank are distinct institutions with very different roles, this continuity implies that the measurement bias on colonial health inputs data, if it exists, is minor.

Health outcomes' levels can be compared to country-level statistics provided by the World Health Organization. This can be done for the number of smallpox casualties for Benin, Burkina Faso, Guinea, Niger, and Senegal.[11] Figure 1.4 shows the number of smallpox casualties according to colonial data, from 1910 to 1958 (plain lines), and according to WHO data, from 1958 to 1969 (dashed lines). The conclusion is not as straightforward as with health inputs,

[11] Unfortunately, data are not available for Cote d'Ivoire, Mali, and Mauritania.

which is not surprising as diseases' casualties are much more volatile than health inputs. Yet, orders of magnitude are coherent between colonial and post-colonial series.

**Figure 1.4:** Smallpox casualties per 1,000 c.: colonial vs World Health Organization data




Author's own computation: dashed lines and dots are extracted from World Health Organization data (only one point in time for Burkina Faso), plain lines are extracted from colonial data used in this paper.

## 1.4 Colonial policies: first descriptive evidence

### 1.4.1 Colonial health policies: what was done?

Table A-1.1 in the Appendix, describes colonial health policies that were implemented in former French West Africa from 1904 to 1958, according to administrative reports available. Five periods are considered. For each of these periods, the table shows what was done in the following five categories of health policies:

1. Treated diseases: diseases for which people were treated, hospitalized, etc.

2. Surveyed diseases: diseases for which the number of casualties was counted.

3. Surveyed deaths: diseases for which the number of fatalities was counted.

4. Vaccinations: diseases for which a vaccine was made available.

5. Other: information on abortions, deliveries, consultations related to pregnancies and school prevention.

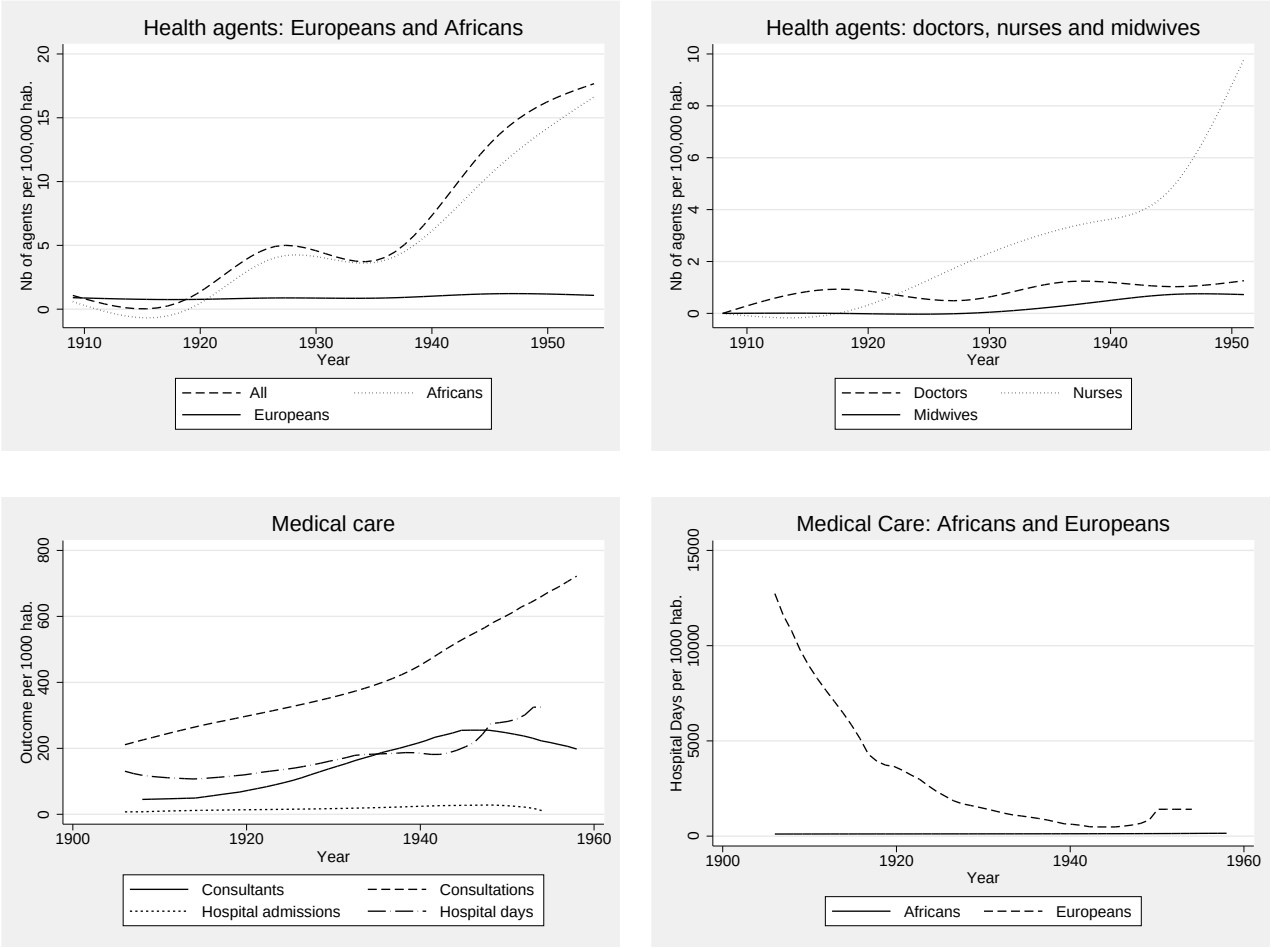
For each period and colony, there is systematic information on medical staff, consultations and consultants, admissions and length of stay in hospital. For a matter of space, these categories are not in the table but they would appear in each table cell.

The first thing that strikingly comes out from this analysis is that much more health services were provided in Senegal than in other colonies. More generally, health policies seem to follow countries where the European presence is greater. The assumption that more health policies are implemented in places where there are more Europeans is tested in Section 1.4.5.

Regarding vaccinations, the main efforts are put on smallpox eradication all over the period. Tuberculosis vaccinations do not start before the 1950's, except in Dakar.[12] Starting then, however, the fight against tuberculosis plays a key role in colonial health policies. From the mid-1930's, leprosy treatments are implemented in every colony, following the building of an *Institut Social de la Lèpre* in Bamako in 1935. There is not much insight into plague prophylaxis: vaccinations are only observed in Senegal from 1928 to 1954, in Guinea in 1954 and in Mali in 1958; and the disease was only surveyed in Senegal and in Guinea during the 1950's.

### 1.4.2 Increasing trends in colonial investments

**Figure 1.5:** Trends in health provision per current population




Trends are obtained by graphing smoothed values of kernel-weighted local polynomial regression of health provision variables on time. For medical staff only, cross medians are calculated and then used as knots to fit a cubic spline, in order to get rid of outliers. Health provision variables are divided by the relevant current population: total population in most cases, African or European population when variables are disaggregated according to these categories.

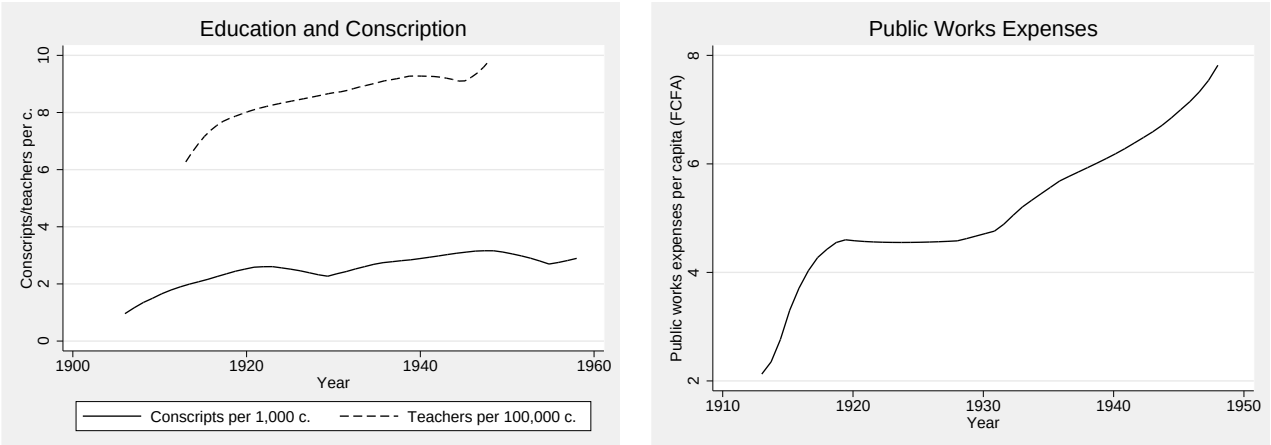
Figure 1.5 shows huge increases in medical staff, mainly driven by the increase in African staff (upper-left graph). According to Becker and Collignon (1998), most of the doctors were

[12] Tuberculosis vaccinations started as early as 1924 in Dakar (Boisseau and Vauguel, 1931). The decision was taken by the French Minister of Colonies, who decided that laboratories of Dakar, Saigon and Antananarivo should start vaccinating infants and conscripts, at a time when vaccination was still very scarce in France.

military doctors in the beginning of the period. Hence, the proportion of European doctors was very high. Then, the ratio African staff / European staff significantly increased over time. Lasker (1977) indeed explains that at least in the context of Cote d'Ivoire, as "the demand for medical services for both Europeans and Africans increases, French doctors were increasingly supplemented by African personnel". Furthermore, health services are moving towards more basic health care, delivered by nurses rather than doctors (upper-right graph). Medical staff provision really starts to increase in the 1920's, following Sarraut's call for the construction of health centers and maternity centers in Africa and for the need to increase the number of staff: "the necessity, in a word, to conserve and increase the human capital in order to make money capital work profitably" (1923). In Figure 1.5 again, medical care – as measured by admissions, length of stay in the hospital, consultants, and consultations – increased significantly over the period (lower-left graph). This increase is driven by the African demand rather than the European one (lower-right graph).

Trends in health provision can be compared to trends in other types of colonial investments. The left graph of Figure 1.6 displays smoothed trends of the number of teachers and of the number of conscripts. The right graph shows the trend of public works' expenses. The picture is similar to health provision: these colonial inputs increase over the period.

**Figure 1.6:** Trends in other colonial investments per current population




Kernel-weighted local polynomial regressions, see Figure 1.5. The public works' expenses trend must be interpreted with caution because of inflation.

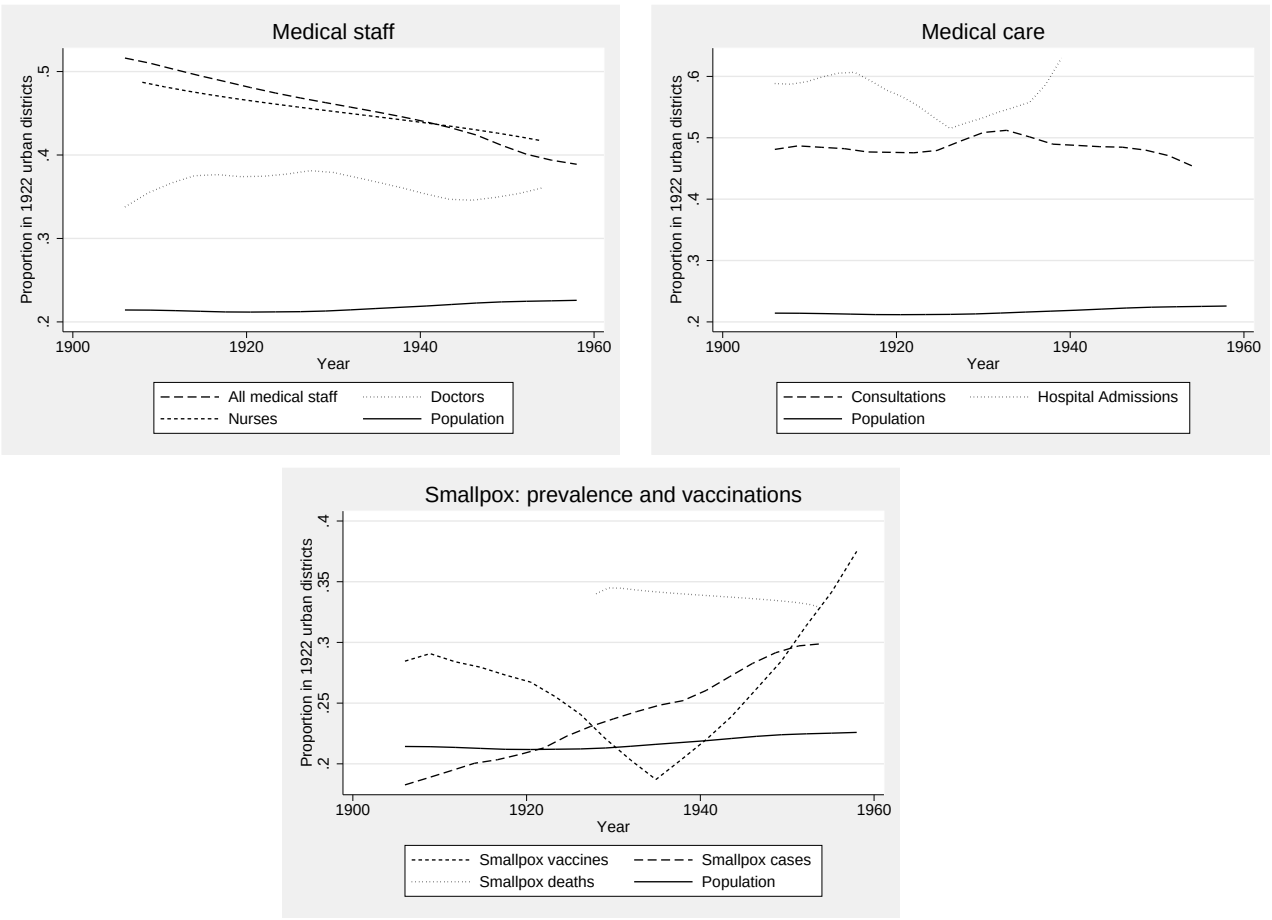
### 1.4.3 Health provision across space

Figure A-1.3 in the Appendix shows the map of the number of all types of medical staff (nurses, midwives, physicians, medical assistants) per 100,000 capita, per district and period. One can observe huge inequalities between districts, with some districts getting less than 5 medical agents per 100,000 capita, while others get more than 15 agents. Similarly for other types of colonial investments, inputs are not evenly distributed across districts.

If health services are unequally distributed, who are they targeting? Is Feierman (1985) right in saying that "the continent's rulers provided medical services to the cities but not the countryside, to men but not to women and children, and to the rich but not the poor"? Are health policies targeting specific populations in the same way as other colonial investments?

### 1.4.4 Colonial policies and urbanization

**Figure 1.7:** One quarter of the population lives in early-urbanized districts: What proportion of health provision do they get?



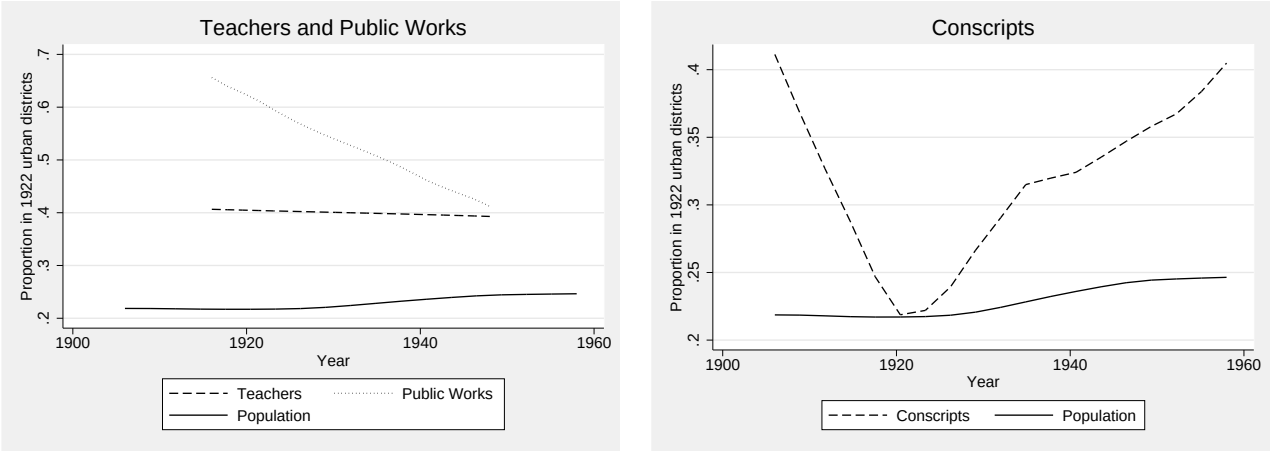

Dashed and dotted graphs show kernel-weighted local polynomial smoothing of health input variables on time in early-urbanized districts. The plain line shows the evolution of population in these same districts. Early-urbanized districts: districts that were already urbanized in 1922.

The first intuition to be tested is whether health provision favors urban districts. According to Echenberg (2002), one of the early strategies of the colonial administration was to put a strong emphasis on urban centers, where the French lived. Becker and Collignon (1998) further state that "health structures were essentially developed in urban areas, where they were serving the European population". To test for this assumption, "early-urbanized" districts are defined as districts that were already urbanized in 1922. They concentrate around one quarter of the population: how much health provision do they get? Figure 1.7 provides evidence that there is a very unequal repartition of medical staff between early-urbanized districts and others. These

districts get between 40 and 45% of medical staff even though they only concentrate between 25 and 30% of the population (upper-left graph). The allocation is even more unequal for less-qualified agents: in the beginning of the period, early-urbanized districts get more than 60% of nurses. However, while the proportion of population living in these districts slowly increases over time, the share of medical staff provision they get decreases (this is not true for doctors), so that inequalities between these districts decrease. This result could be driven by the fact that other districts are now urbanizing. Early-urbanized districts also get around one half of medical care, and this share is not significantly decreasing over the period (upper-right graph of Figure 1.7).

These specific districts do get more health provision, but what about epidemics? Could it be the case that they indeed *need* more health provision? The bottom graph of Figure 1.7 shows that early-urbanized districts get a little more smallpox vaccinations at the end of the period. Generally speaking, they do not get proportionally more vaccinations all over the period. That is despite the fact that the reported disease prevalence is higher in these districts: they concentrate around 35% of reported smallpox deaths. While early-urbanized districts face a higher disease prevalence, they only start receiving relatively more smallpox vaccines per capita at the end of the colonial period. Yet, the monitoring of disease is probably higher in cities, which would bias their reporting upward. Furthermore, early-urbanized districts are very much favored in terms of medical staff and medical care allocation. The number of vaccinations per medical staff is thus lower in these districts. They focus more on curative than preventive health care, relatively to other districts.

**Figure 1.8:** One quarter of the population lives in early-urbanized districts: What proportion of other colonial policies do they get?




See Figure 1.7.

As a comparison, Figure 1.8 looks at other types of colonial inputs: what proportion of these inputs do early-urbanized districts get? These districts also get disproportionably more teachers and public works than others. They also concentrate a higher share of conscripts, apart from the early 1920's. All in all, early-urbanized districts get systematically more colonial investments for all types of policies, all over the period. The repartition is less and less unequal over time for medical staff and public works only.

### 1.4.5 Colonial policies and Europeans

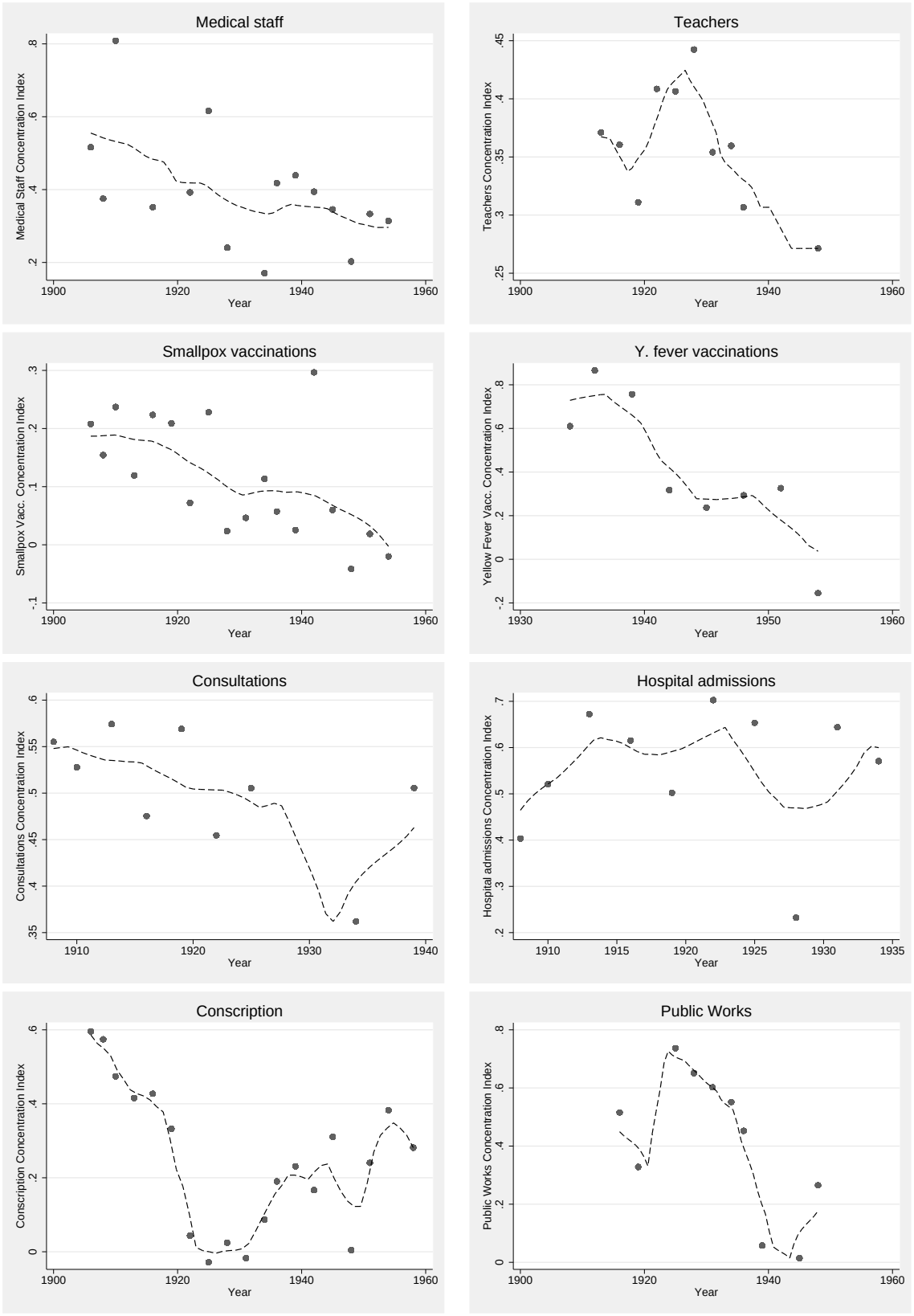
The second intuition which is tested here is whether colonial policies target districts where Europeans live. Regarding health, during the first years of colonization in Cote d'Ivoire, "small medical posts began to appear at the major administrative and military centers of the interior, wherever there was a European settlement and economic interests to protect" (Lasker, 1977). Concentration indexes are used to test for this assumption, following Kakwani, A.Wagstaff, and van Doorslaer (1997). Let $X_i$ (i = 1...N) be the level of colonial inputs (medical staff, vaccinations, teachers, etc, per current population) of the $i^{\text{th}}$ district. From there, $R_i^{eur}$ is the relative rank of the $i^{\text{th}}$ district regarding European presence. The concentration index $C^{eur}$ is defined as :

$$C^{eur} = \frac{2}{N\mu} \sum_{i=1}^{N} X_i R_i^{eur} - 1$$

Where $\mu = \sum_{i=1}^{N} X_i$ is the mean level of colonial investments. $C^{eur}$ thus indicates how much colonial inputs are concentrated on observations with high ranks in Europeans' proportion. If X is independent from European density, then $C^{eur} = 0$.

Concentration indexes are computed for each period and each colonial input variable and are graphed in Figure 1.9. These graphs show that concentration is well above zero for every dimension of colonial policies. For most dimensions of colonial investments, there are high but decreasing concentration indexes. The bias towards Europeans is not significantly decreasing for hospital admission and only slowly decreasing for consultations. Indexes are significantly positive for both smallpox and yellow fever vaccinations; but they are much higher for yellow fever vaccinations. For both diseases, indexes decrease a lot and become negative at the end of the period: vaccinations focus on non-European districts. The same pattern is true for public works, which get to null concentration indexes at the end of the period.

**Figure 1.9:** The concentration of colonial investments towards europeanized districts




Graphs show scatter plot, together with kernel-weighted local polynomial smoothing, of concentration indexes on time. Concentration indexes indicate how much health provision is concentrated on observations with high ranks in Europeans relative population.

To conclude, there is a big increase in colonial investments over the period. The positive trend in health provision is driven by a greater increase in health care provided by African staff and nurses, outside of hospitals. There is also a great heterogeneity of colonial services' provision across districts. This heterogeneity is partly driven by the fact that early urbanized districts get proportionally more investments and that there is a high concentration of inputs' provision towards Europeans. Heterogeneity decreases over time, apart from hospital-related health care (doctors, hospital admissions, etc.) and military expenses.

## 1.5 Health policies and other colonial policies: Context and empirical strategy

The first assumption of this work is that due to the scarcity of resources, all colonial expenses studied here – education, health, public works, conscription – [13] were bound by budgetary constraints in former French West Africa. Consequently, for each category of colonial policies, some choices had to be made regarding the allocation of budget across policies, and across districts. Regarding health policies, the colonial power needed to decide what was produced in the health system, and which districts were targeted. This section describes the context of health provision's decisions, within colonial policies' decisions. It discusses various potential scenarii regarding the allocation of colonial policies, and their implications. The empirical part will allow to rule out some of these scenarii and to identify the drivers of health policies' allocation, in comparison to other colonial policies.

### 1.5.1 The decision-making chain of colonial investments

At each period $t$, the decision was taken to spend an amount $N$ on the four types of colonial expenses considered here: conscription, education, health, and public works. A part $P_h$ of $N$ was then allocated to current health expenses in colonial French West Africa. At the same time, a decision could be taken to contract a loan in order to finance an important health infrastructure investment, such as a hospital building. The General Governor of former French West Africa in Dakar was then in charge of dividing the amount $P_h.N$ between the eight colonies.

At this point, the governor of each colony did some budget planning.[14] The budget was shared between two types of spending: staff and equipment. Within each of these expenses, the budget could be further shared between various medical services: head of medical services and pharmacy, hospitals, Native Medical Assistance, vaccination services, etc. In the few evidence provided in the archives, the Native Medical Assistance budget amounted to around half of

[13] These are the four types of colonial expenses studied in this paper. I do not claim that these were the only expenses made by the colonial power.

[14] For some colony×year, colonial archives provide suggestive evidence of this.

the total budget. The amount dedicated to the Native Medical Assistance was shared between colonial districts;[15] and followed a "delegation" of decision from the colony governor to district governors. These district governors received a budget allowance for the local Native Medical Assistance, which they could allocate to various health inputs.

As a consequence, the final quantity of inputs provided in a given district and period was determined by a whole decision-making chain. It depended on the amount $N$ allocated to colonial expenses, on the share of the budget that was devoted to each category of policy (health, education, conscription and public works), and then on the geographical allocation across space. The outcome of this decision-making chain is that each district $z$ gets a quantity of investment $I_{z,t}$ in period $t$. This paper considers health as multidimensional. It tries to explain the provision of various health inputs: physicians, African staff, vaccines, etc. The main aim of this work is to describe the determinants of colonial health investments, as compared to other colonial policies. What are the underlying mechanisms leading to the allocation of health provision? To what extent are they similar to the mechanisms underlying other colonial policies? What kind of strategy did the colonial power implement?

In order to describe colonial policies' drivers, a theoretical distinction can be made between two types of decisions. First, a binary "extensive investment" decision, by which the colonial administration decides to increase the number of inputs from $0$ to $y > 0$ in district $z$. Second, an "extensive investment" decision, by which the number of inputs is decided, once it has already been decided to allocate a positive number of inputs. As discussed in the next section, the underlying mechanisms leading to these two types of decisions may not be the same. This distinction will sometimes allow to disentangle between various possible channels. Section 1.5.3 presents an empirical model which allows to make the distinction between the decision to go from zero to a positive number of inputs, and the decision to increase the number of inputs.

### 1.5.2 The provision of colonial policies: what are the main drivers?

The first set of determinants for colonial policies are time and geography. Colonial administrations' decisions on investments varied a lot across time. As shown by descriptive statistics, there were increasing trends in colonial investments over the period. Furthermore, the target of colonial policies also changed over time (health policies aimed at curative, and then preventive action, for instance). As a result, the year $t$ is expected to be a determinant of $I_{z,t}$. Also, each district has a preexisting colonial system, pre-colonial institutions, geographic patterns, etc. All of these districts' characteristics have an impact on the provision of colonial inputs. This pa-

[15] Table A-1.2 in the Appendix provides examples of budget delegations to districts in three colony×year. Unfortunately, such detailed data is only available for a couple of observations. This descriptive evidence shows that budget was not directly proportional to population, nor exactly equal in each districts. The allocation rule was more complicated than a fixed amount per capita or per district.

per seeks to describe the local drivers of the colonial system's development in former French West Africa. It thus focuses on colonial policies' determinants other than fixed geographic or periodic characteristics.

The second set of determinants for colonial investments are preferences. The colonial administration can chose to provide a preferential treatment to some given districts, or to some specific populations. It can also chose to have a specific target in mind regarding the dynamic allocation of colonial policies.

The third set of determinants are related to the cost and benefit of colonial policies. It is indeed very likely that a decisive aim of colonial investments was their productivity. Hence, "the allocation of health inputs outside of urban centers was determined by economic criteria (productivity, cost, etc)" (Becker and Collignon, 1998). Increased productivity can be an intrinsic motivation if the colonial power has a philanthropic goal. The productivity aim can also result from an economic incentive. For health and education policies, there is an incentive to provide a healthy and educated workforce. Dozon (1985) explains that from the 1930's, the economic interest of colonies is put forward and thus becomes the main incentive to provide health services. For public works and conscription, the immediate interests of the colonial power are even more obvious. Furthermore, one can think of a "propaganda" motivation by which the colonial power wants to advertise the success of its colonial policies, notably in France. Either of these motivations – and the list is probably not exhaustive – would make the productivity of colonial policies an essential determinant of colonial services' provision.

Under this assumption, the colonial administration was running a cost-benefit analysis to allocate colonial policies, given its preferences. From descriptive results, two extreme case scenarii can already be ruled out. The allocation of colonial policies was neither random, nor perfectly even across districts. In the first case, none of the potential determinants tested in this work would be robustly significant. In the latter case, each district would get the same quantity of investments per capita, which is far from being the case. Away from these extreme cases, what were the main drivers underlying the allocation of policies? What were the potential scenarii for the allocation of colonial investments? Were there specificities in the allocation according to the type of colonial policies?

**The colonial system**

Colonial investments' decisions are taken within an existing colonial system. For each period, the colonial system can be described as a set of colonial districts. The structure of the colonial system in $t-1$ has an impact on investment decisions made in $t$. It can further be assumed that decisions for $z$ are impacted by existing inputs and policies in $z$, but also in districts close to $z$. Colonial districts can be seen as a network of districts, $G$. Then, for each given

district $z$, a network of neighboring districts $G(z)$ is defined. $G(z)$ contains all neighboring districts of $z$, excluding $z$ itself.[16] The structure of the colonial system in $z$ and $G(z)$ has an impact on investments' decisions, through several mechanisms.

*Returns to scale and path dependence*

How do colonial investments relate to existing colonial infrastructures? Is there a complementarity between new colonial investments and existing infrastructures? There are two main channels which could lead to more investments being made in places which already have colonial infrastructures: a returns to scale channel and a path dependence channel.

First, under the plausible assumption that there is a fixed cost to colonial investments, inputs complementarity can rise returns to investments. In this context, it is more costly to bring vaccines or to establish medical staff or teachers in places that are more remote within the colonial system. For instance, it is more costly to provide health policies in places where there are no existing medical facilities, no history of vaccinations, etc. Second, the colonial administration could have an intrinsic preference for some districts, in which case these districts would receive a preferential treatment at each period, leading to path dependence. In this scenario, colonial investments would also increase with colonial infrastructure.

If fixed cost is the main channel, the complementarity of inputs mainly matters for the binary decision to start investing in a district: it should matter more for the extensive than for the intensive margin of investments. However, if the main channel is the persistence of health inputs' repartition through path dependence, it should matter more for intensive investment decisions.

A further question is whether the principle of investments' complementarity is general – the presence of a school decreases the cost of vaccination – or rather specific – the presence of a school decreases the cost of teachers only.

*"Coverage" and "diversification"*

An antagonist scenario is related to the administration's preferences.

It could be the case that the colonial administration has a strong preference to maximize its "coverage". During the first part of the colonial period in particular, the colonial power seeks to occupy the land that was recently conquered through "coverage". One way to cover colonial territories is to establish a local colonial administration and to deliver services locally. Potentially, all types of colonial policies may be used as a tool for "coverage". Under such

[16] All neighbors, within and outside the colony, are included. Results are robust to keeping only within-colony neighbors. However, it seemed more relevant to include all neighbors, since countries' borders were still very porous and therefore epidemics did not stop at borders.

a logic, there is a higher probability that investments are made in places where the colonial administration is not very much present (intensive, rather than extensive margin). Two sorts of "coverage" could be considered. First, a "general colonial coverage": the administration seeks to develop at least one colonial service in each district. Its first aim is the colonial presence, whatever the kind of presence. In this scenario, the probability that colonial investments are made decreases with all types of colonial presence. Second, a "specific colonial coverage", by which the administration wants both teachers and medical staff in each district, for instance. In this scenario, the probability of colonial policies decreases with same-type colonial presence.

Another scenario, which could be compatible with "coverage", is that the colonial administration seeks "diversification". That is the case if the *quantity* of investments decreases with colonial presence, or with the lagged stock of inputs. "Diversification" could be driven by a colonial administration's preference for an homogenization of districts' provision. It could also be explained by the fact that some investments have long-lasting effects. The latter mechanism would be more plausible for health and public works' expenses.

These two scenarii do not exclude each other. It could be the case that the colonial administration makes a trade-off between cost reduction and "coverage". For instance, the probability of investment could be higher in presence of one given infrastructure, and at the same time more investments could be made in districts which were not allocated with one other input yet.

**Context: do investments target specific populations?**

A second mechanism tested in this work is the targeting of specific populations. Indeed, following descriptive statistics, one straightforward question is whether colonial policies target places with a higher proportion of urban or European population, all things being equal. Descriptively, urbanized and europeanized districts get more colonial policies. The reason for that could be that urban districts are also districts where disease prevalence is higher. It could also be the case that urban districts get more investments to start with, and are thus more targeted afterwards because of path dependence. Consequently, one research question is: given the structure of the existing colonial system, and the disease prevalence in the district, are urban districts more targeted by colonial policies? A second question is: are health policies more or less biased towards these populations than other types of colonial policies? If, all things being equal, specific populations are targeted, is it possible to identify the main mechanism underlying this targeting?

There are two potential mechanisms tested in this paper: a preference channel and a cost channel. First, the colonial power could have an intrinsic motivation to favor these popula-

tions if it places a greater interest in their well-being. If this was the main mechanism, the population context would not matter differently according to the type of decision - intensive or extensive. Rather, it could matter differently depending of the type of colonial policies. For instance, making the analogy with Costa and Kahn's self-interest of the American middle class, the Europeans probably had a further motivating factor to provide better health in europeanized districts. Indeed, they could have had a self-interest to prevent epidemics among African societies, in order to protect themselves from these diseases. This preference channel would probably have a greater impact on health than on education investments.

Second, there might be some increasing returns to population for investments, which are linked to investments' fixed costs, for instance related to transportation, facility building, etc. In that case, it is less costly to invest in urban areas. If this second mechanism prevails, the presence of cities would matter for all types of colonial investments.

**The prevalence of diseases**

Does the prevalence of diseases matter for the allocation of colonial policies? Do contagious and non-contagious diseases have the same impact? What are the possible scenarii regarding these drivers?

First, the colonial power could have an incentive to provide colonial policies in places where the supply will be more surely met, i.e. where the demand for colonial policies is higher. The past recorded number of diseases' casualties could be interpreted as a degree of "sympathy" towards the colonial presence, or at least towards colonial policies. Allocating policies to such districts would rise the productivity of colonial investments.

Second, in terms of health-related policies, the colonial administration could have a higher benefit to target places where investments are most needed. This means targeting places where the contagion risk is higher, or where health needs are greater, which are both captured by disease prevalence. According to Lasker (1977), "preventive health care through vaccination and hygiene campaigns aimed at reducing the epidemics which had such a murderous effect on the French". Becker and Collignon (1998) also insist that "the African medical assistance was developed in rural areas, mainly to stop epidemics".

The demand channel is expected to have a greater impact on the extensive margin of investments. The colonial administration starts investing in places where it believes that the supply would be met. However, there is no good reason why the quantity of investments would be determined by this imperfect measure of colonial policies' demand. Furthermore, this channel could matter for various dimensions of colonial policies, not only health. On the contrary, health needs should matter for both the intensive and extensive margin of health investments and they should matter for health-related inputs only.

### 1.5.3 The empirical strategy

#### Basic model

The basic model which allows to test for these possible scenarii is the following:

$$I_{z,t} = \alpha + \beta.System_{z,t-1} + \gamma.Context_{z,t-1} + \delta.Diseases_{z,t-1} + \lambda_t + \mu_z + \epsilon_{zt} \quad (1.1)$$

$I_{z,t}$ is the measure of colonial inputs in district $z$ and period $t$. The model contains period and district dummies to absorb fixed geographic and time determinants. The model also contains three vectors of explanatory variables to test for the theoretical determinants of colonial policies. All these variables are added in lags in the model, to avoid reverse causality.

$I_{z,t}$ variables are converted into population ratios: vaccinations and conscripts are per 1,000 capita (as of 1925), staff (health and education) are per 100,000 capita (as of 1925), public works are in FCFA per capita.[17] All equations are weighed by a ratio of district population over all former French West African population in the given period. This gives a higher weight to more populated districts, and gives the same weight to each period (otherwise, a bigger weight would be artificially given to more recent periods). Standard errors are clustered at the district level, to correct for autocorrelation of unobservables.

*The colonial system*

Four variables are built to describe the existing colonial system in the previous period. The first variable describes the logistical support for medical care: an indicator variable of whether there was a medical center is built.[18] Second, the structure of the colonial system is described by an index of the quantity of colonial staff in district $z$ and its neighbors $G(z)$.[19] Third, two dummy variables describe the presence of the colonial administration in district $z$ and its neighbors. The dummy "strong presence of health administrations in $t-1$" is equal to one if there are at least three positive stocks for the following health inputs: vaccinations in $z$, medical staff in $z$, vaccinations in $G(z)$ and medical staff in $G(z)$. The dummy "strong presence of other administrations in $t-1$" is equal to one if three of these colonial inputs' stocks are positive in $t-1$: teachers in $z$, public works in $z$, conscription in $z$, teachers in $G(z)$, public works in $G(z)$ and conscription in $G(z)$.[20]

[17] Ratios are for 1925 population of district $z$, rather than current population. As current population changes over time, it could induce a spurious negative correlation between all ratio variables. All discrete explanatory variables in the model are also converted into population ratios, using the same transformation. Furthermore, 1925 population data are used as benchmark data because they are the most reliable.

[18] Colonial data do not provide direct evidence on medical center presence. It is thus assumed that a medical center existed if there is a record of any medical care, other than "outside of dispensaries consultations".

[19] Following descriptive statistics, colonial staff variables are weighed in the following way: one for doctors, one fourth for nurses, one half for other medical staff and one sixth for teachers; $z$ and $G(z)$ are given the same weight.

[20] Two thirds (resp. 60 %) of the observations have a strong presence of health administrations (resp. other

*Context*

The context is captured by a dummy variables for the lag of city presence. This variable is very much correlated with population density and with European presence. Consequently, all variables can not be included, as they would introduce collinearity in the model. Section 1.6.1 provides evidence that it would be equivalent to include in the model urban population, European population, or population density.

*Diseases*

A set of three dummy variables measures the lagged prevalence of diseases in district $z$.[21] The model includes a categorical variable for the *relative* prevalence of contagious diseases in the district.[22] This variable measures what happens if the prevalence of contagious diseases is high (casualties per 1925 capita above the period's median) or low (casualties below the period's median) compared to a null prevalence. Furthermore, the impact of a positive number of non-contagious casualties is tested. By definition, the prevalence of non-contagious diseases is not related to a contagion risk. However, it could measure a demand for health or health needs in the district.

There is a direct reverse causality bias going from health provision to disease prevalence. Casualties numbers could also be endogenous to health provision because of a measurement error bias, or of an observation bias. When health inputs increase, there is more attention towards diseases, diseases are better tracked, leading to a positive bias. These biases are dealt with by using the lagged value of casualties to compute these variables. Furthermore, an important distinction is made between null and missing values. A null number of casualties means that disease casualties were counted and found null, whereas a missing value means that casualties were either not counted, either not reported. The impact of low or high prevalence is compared to a null prevalence, i.e. a setting in which diseases are looked for, but no casualties were found. A dummy for the missing number of casualties is added.

**Two-tier model, Cragg (1971)**

In the specific context of this paper, three outcome variables $I_{z,t}$ – the number of vaccinations, the number of staff and the number of teachers – are left-truncated at zero. Figure 1.10 provides histograms for the distribution of these variables. For these non-negative outcomes, observations pile up at zero. More precisely, 13% of teachers, 20% of smallpox vaccinations

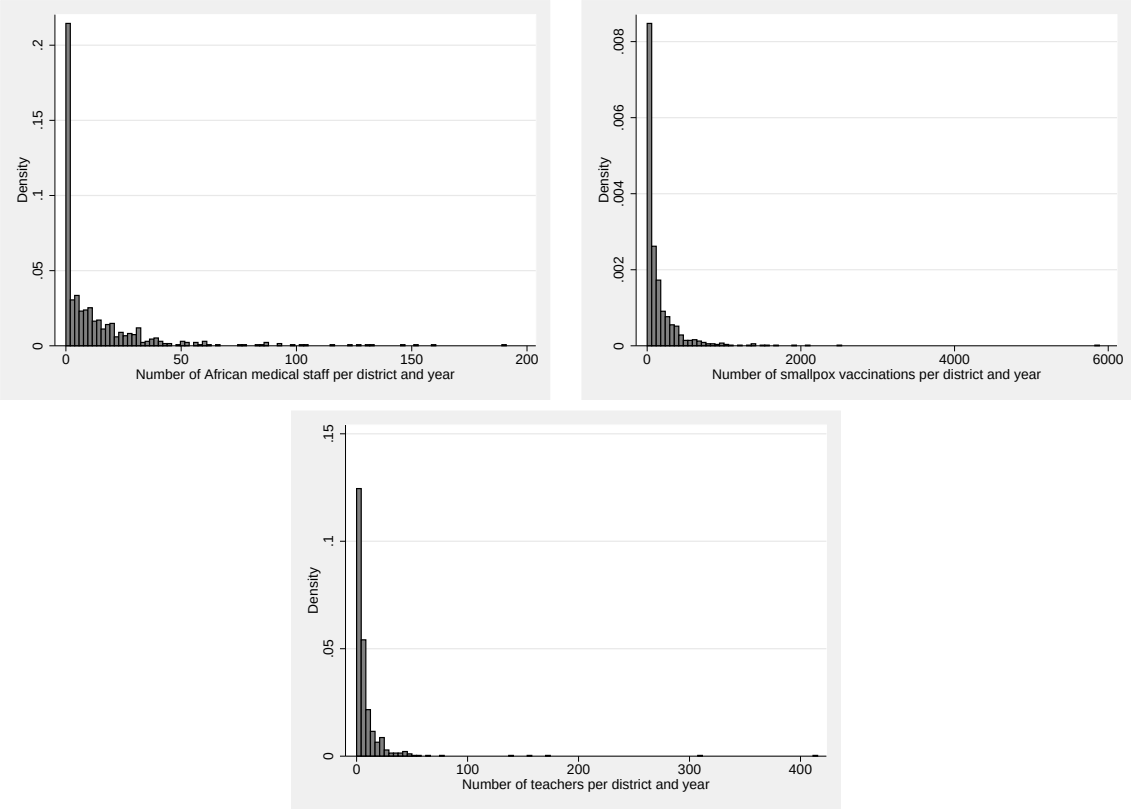
administrations).

[21] Variables for the prevalence of diseases in $G(z)$ could not be included. They are highly correlated with the risk in $z$, at least for contagious diseases, and would introduce collinearity in the model.

[22] Diseases listed as contagious are: canker, chickenpox, diphteria, filariasis, gonorrhoea, malaria, measles, meningitis, mumps, pertussis, plague, pneumonia, polio, smallpox, syphilis, trachoma, trypanosomiasis, yaws and yellow fever.

and 35% of African medical staff are at zero. As a consequence, it may be more relevant to use a model which fits better with limited dependent variables, such as a tobit model. Cragg (1971) proposes a flexible alternative to the tobit model, which is sometimes called a "two-tier" model. This model allows the probability of a positive value and the actual value, given that it is positive, to be determined by separate processes. Such a model fits well with the theory described in Section 1.5.2.

**Figure 1.10:** Left-truncation of outcome variables:
Histograms for African medical staff, smallpox vaccinations and teachers




These figures graph 30 bins' histograms for the distribution of African medical staff, smallpox vaccinations and teachers. Histograms show that the main outcomes used in this paper pile up at zero.

Cragg's model integrates the probit model to determine the probability of the outcome $y$ to be positive, and the truncated normal model for given values of $y$. This gives the following likelihood function:

$$f(w, y|x_1, x_2) = \{1 - \Phi(x_1\gamma)\}^{\mathbb{1}(w=0)}[\Phi(x_1\gamma)(2\pi)^{-\frac{1}{2}}\sigma^{-1} \exp\{-(y - x_2\beta)^2/2\sigma^2\}/\Phi(x_2\beta/\sigma)]^{\mathbb{1}(w=1)} \quad (1.2)$$

Where $w$ is a binary indicator equal to 1 if $y$ is positive and to 0 otherwise. In this model, compared to the tobit model, the probability of $y > 0$ and the value of $y$, given $y > 0$, are determined by different mechanisms (the vectors $\gamma$ and $\beta$, respectively). One reassuring result

regarding the validity of the model is the fact that the estimated unconditional expected value of $y$, from Cragg's model, is very close to results from the general tobit model. However, results presented in Section 1.6.2 imply that $\gamma$ and $\beta/\sigma$ are significantly different, making Cragg's model more relevant to fit the data.[23] From this model, the same probabilities and expected values as with the tobit model can be obtained: the probability that y is positive $P(y_i > 0|x_{1i})$, the expected value of $y$, conditional on $y > 0$, $\mathbb{E}(y_i|y_i > 0, x_{2i})$, and the unconditional expected value of $y$, $\mathbb{E}(y_i|x_{1i}, x_{2i})$.

The user-written STATA command *craggit* is used to fit Cragg's model to the data. Formula provided in Burke (2009) are used to compute marginal effects. Standard errors are approximated using the delta method.

The same set of explanatory variables are used in the first and second tier equations. The only difference between both equations is that the second tier equation controls for district fixed effects, while the first tier equation only controls for colony fixed effects. The decision process which is modeled in the first tier equation makes it irrelevant to add district fixed effects in the estimation. The underlying research questions for the first tier equation is: why does the colonial administration start to provide colonial services in some districts rather than others? The idea is thus to compare districts. On the contrary, the second tier equation studies the determinants of the quantity of colonial inputs in a given district. Hence, as mentioned in Section 1.5, the empirical strategy aims at absorbing any fixed characteristic of the district.

## 1.6 Results: what are the main determinants of health provision ?

### 1.6.1 Linear results

Table 1.1 reports estimation results of Equation (1.1), using respectively African medical staff, physicians,[24] smallpox vaccinations, teachers, enlisted conscripts and public works (in FCFA) as endogenous variables. Baseline statistics (Table A-1.3 in the Appendix), and mean values of inputs (bottom of Table 1.1) can be used to interpret the magnitude of the effects.

Moreover, as Section 1.4 shows that the type and the quantity of investments change dramatically across different periods, I want to look at the heterogeneity of the effects across time. Historians' works and descriptive evidence provided in this paper generally identify World War 2 as a turning point in colonial investments. From that point on, colonial investments increase dramatically in many fields: health, education, infrastructure, etc. Two main periods are identified to explain the main endogenous variables. For African medical staff, the sample is

[23] Tobit results are available on request.

[24] This first set of results makes the distinction between physicians and African medical staff. In the following of the paper, only African medical staff will be used as endogenous variables. Looking at African medical staff rather than physicians reduces the risk of reverse causality when using the number of Europeans as an explanatory variable, which could go through the hiring of European staff.

cut in half, in 1943. As for other types of colonial policies a lot of investments happened in the beginning of the colonial period, cutting the sample in the middle of World War 2 would lead to a very small number of observations in the second period. Consequently, for other colonial inputs, the chosen cutting point is 1932. Estimation results across periods are shown in the Appendix, in Table A-1.4 for health policies, and in Table A-1.5 for other policies.

The first general result from Table 1.1 is that there is a positive correlation between colonial expenses' trends. The number of medical staff and of conscripts increases with colonial staff in $z$ and $G(z)$. For one more colonial staff (amounting to one more doctor, or four more nurses, or six more teachers), a district gets around $0.2$ more medical staff and enlists $0.26$ more conscripts. These effects mainly come from the late colonial period.[25] Trends in the number of teachers and in the number of smallpox vaccinations also correlate to colonial staff's trends in the first period. Before 1932, one more colonial staff leads to two more vaccinations, and $0.06$ more teachers. Moreover, in the late colonial period, there are eleven more teachers in districts with a strong presence of other administrations in the previous period.

Another finding is that health investments increase with the presence of a medical center in region $z$ in the previous period. This result is specific to health investments; there are no returns to scale of medical center's presence for other types of investments.

Taken together, these results point towards a complementarity of colonial inputs, which could be interpreted for now as a preference for path dependence, or as the result of returns to scale. The only contradicting finding to these results is the fact that there are more than seven less African medical staff allocated to the district if there was a strong presence of other administrations in the previous period, which would rather points towards a "coverage" strategy.

Another significant result is that some dimensions of colonial investments increase with disease prevalence. More precisely, there are around eight more African medical staff and two more conscripts if non-contagious prevalence was positive in $t-1$. From 1932, public work's expenses are also greater in districts with a positive prevalence of non-contagious diseases.

In the second period only, smallpox vaccinations target places where the observed contagious prevalence was higher. Compared to places with no casualties from contagious diseases in the previous period, districts with a relatively low prevalence get $87$ more vaccines, and districts with a relatively high prevalence get $170$ more vaccines. The fact that prevalence does not matter for vaccinations' allocation in the first period is in line with Dozon (1985), who explains that the Native Medical Assistance was originally aiming at healing individuals, before focusing on the prevention of diseases and epidemics.

[25] See Tables A-1.4 and A-1.5 in the Appendix.

**Table 1.1:** Colonial inputs' determinants

| | Colonial Investments | | | | | |
|---|---|---|---|---|---|---|
| | **African Med. Staff** | **Physicians** | **Smallpox Vaccinations** | **Teachers** | **Conscripts** | **Public Works** |
| | per 100,000 hab. | | per 1,000 hab. | | | per hab. |
| **The structure of the colonial system:** | | | | | | |
| $\mathbb{1}\{\text{Medical center in } t-1>0\}$ | 3.359 | 1.551*** | 31.971* | -0.801 | -0.448 | 0.502 |
| | (2.978) | (0.579) | (18.021) | (0.904) | (0.318) | (1.186) |
| Colonial staff in $t-1$ | 0.222*** | 0.190*** | 0.146 | 0.068 | 0.262*** | 0.048 |
| | (0.081) | (0.055) | (0.510) | (0.047) | (0.047) | (0.043) |
| Strong presence of health adm. in $t-1$ | 3.326 | -1.780 | -31.856 | 0.868 | -0.028 | -1.521 |
| | (2.452) | (1.356) | (35.133) | (0.905) | (0.513) | (1.018) |
| Strong presence of other adm. in $t-1$ | -7.531* | -1.140 | 13.999 | 1.120** | -0.466 | -1.759 |
| | (4.356) | (0.911) | (28.650) | (0.563) | (1.545) | (1.559) |
| **Context:** | | | | | | |
| $\mathbb{1}\{\text{Cities in } t-1\}$ | 8.406** | 0.864* | 54.207** | 1.693** | 0.005 | 2.649* |
| | (3.863) | (0.507) | (25.145) | (0.781) | (0.667) | (1.562) |
| **Diseases:** | | | | | | |
| $\mathbb{1}\{\text{Contagious diseases in } t-1>0\}$ – low prevalence | 0.492 | 0.553 | 27.473 | 1.085 | -1.552 | 1.037 |
| | (2.051) | (0.338) | (22.031) | (0.996) | (1.030) | (1.294) |
| $\mathbb{1}\{\text{Contagious diseases in } t-1>0\}$ – high prevalence | -2.181 | 1.017 | 60.783* | 0.858 | -1.330 | 0.789 |
| | (2.626) | (0.737) | (33.495) | (0.943) | (1.210) | (1.730) |
| $\mathbb{1}\{\text{Non-contagious diseases in } t-1>0\}$ | 8.407*** | 0.293 | -2.369 | -0.408 | 1.644* | 2.021 |
| | (2.857) | (0.738) | (16.703) | (0.753) | (0.884) | (1.326) |
| R-squared | 0.261 | 0.403 | 0.135 | 0.231 | 0.344 | 0.237 |
| Observations | 697 | 758 | 952 | 662 | 1618 | 394 |
| District and Period F.E. | Yes | Yes | Yes | Yes | Yes | Yes |
| Mean investment | 12.42 | 2.21 | 121.80 | 5.40 | 3.27 | 3.96 |

*** p<0.01, ** p<0.05, * p<0.1. Standard errors clustered at the district level. Data are weighed at the district level. When an explanatory variable contains missing values, a dummy variable for missing values is added. A few outlier observations are dropped from the estimation sample: African medical staff per 100,000 1925 capita $> 300$; smallpox vaccinations per 1,000 1925 capita $> 2500$; and teachers per 100,000 1925 capita $> 100$. The first year of data is dropped from the sample, as lagged of explanatory variables are not available for this year. Colonial staff is the lagged weighted sum of teachers and medical staff in $z$ and $G(z)$ (see text for details). $\mathbb{1}\{\text{Strong presence of health administrations in } t-1\}$=1 if there are at least three positive stocks for the following health inputs: vaccinations in $z$, medical staff in $z$, vaccination in $G(z)$ and medical staff in $G(z)$. $\mathbb{1}\{\text{Strong presence of other administrations in } t-1\}$=1 if the district had at least three positive stocks for the following colonial inputs: teachers in $z$, public works in $z$, conscription in $z$, teachers in $G(z)$, public works in $G(z)$ and conscription in $G(z)$. Low (resp. high) prevalence means that casualties are below (resp. above) the period's median number of casualties, i.e. prevalence is low (resp. high) compared to other districts.

**Table 1.2:** Three drivers: urban population, European population and population density

| | (1) | (2) | (3) | (4) | (5) |
|---|---|---|---|---|---|
| | | $I_{z,t}$: **African medical staff** per 100,000 hab. | | | |
| $\mathbb{1}${Cities in $t-1$} | 8.406** | | | 7.047** | 6.135 |
| | (3.863) | | | (3.541) | (3.886) |
| N Europeans in $t-1$ | | 1.389*** | | 0.889 | |
| | | (0.085) | | (1.020) | |
| Population density in $t-1$ | | | 0.095*** | | -0.104 |
| | | | (0.005) | | (0.346) |
| $\mathbb{1}$ {Cities}×Population density in $t-1$ | | | | | 0.198 |
| | | | | | (0.346) |
| $\mathbb{1}$ {Cities}×N Europeans in $t-1$ | | | | 0.484 | |
| | | | | (1.020) | |
| Observations | 697 | 697 | 697 | 697 | 697 |
| | | $I_{z,t}$: **Smallpox vaccinations** per 1,000 hab. | | | |
| $\mathbb{1}${Cities in $t-1$} | 54.207** | | | 41.680 | 82.520** |
| | (25.145) | | | (25.530) | (37.020) |
| N Europeans in $t-1$ | | 3.273*** | | -4.668 | |
| | | (0.491) | | (3.858) | |
| Population density in $t-1$ | | | 0.226*** | | 2.753 |
| | | | (0.065) | | (1.930) |
| $\mathbb{1}$ {Cities}×Population density in $t-1$ | | | | | -2.515 |
| | | | | | (1.913) |
| $\mathbb{1}$ {Cities}×N Europeans in $t-1$ | | | | 7.838** | |
| | | | | (3.936) | |
| Observations | 952 | 952 | 952 | 952 | 952 |

*** p<0.01, ** p<0.05, * p<0.1. Standard errors clustered at the district level. Data are weighed at the district level. N Europeans: number of Europeans per 1925 $1,000$ hab. Same specification and controls as in Table 1.1. The estimates of a subset of variables are shown.

The last striking result is that, outside of military expenses, all colonial inputs are positively correlated to the presence of a city. As mentioned earlier, the presence of a city is correlated to European population and to population density. Table 1.2 estimates Equation (1.1) in column (1). Then the dummy $\mathbb{1}$\{Cities in $t-1$\} is replaced by the number of Europeans per capita in $t-1$ (column (2)), by population density in $t-1$ (column (3)) and by the interaction of the latter two variables with city presence in columns (4) and (5). Health inputs are biased towards urban and European centers, which are also more densely populated districts.

This is not a health investments' specificity; the same can be shown for education and public works.[26] The ratio medical to all colonial staff is not correlated to urban and European populations. The magnitude of effects are quite comparable for all types of colonial policies: the presence of a city increases the number of teachers by 31%, the number of African medical staff by 66% and the expenses in public works by 68%, compared to mean values.

The presence of a city matters more in the beginning of the period, in terms of magnitude. For instance, the presence of a city is associated with 100% more African medical staff in the first period, versus 55% in the second period, compared to the period's mean. This result points to returns to population, as it is likely that investments' fixed costs are higher in the beginning of the colonial period, when the colonial system is less developed.

[26] Results are available on request.

Estimation results of Equation (1.1) imply that the mechanisms underlying the allocation of health policies are not really specific, compared to other colonial policies. For all types of colonial investments, there is a complementarity in colonial inputs, and the presence of a city is a strong predictor of investments. The prevalence of non-contagious diseases correlates to the allocation of staff, conscripts and public works. The only two specificities are the fact that *(i)* there is a negative correlation between medical staff's allocation and other administrations' presence; *(ii)* the number of smallpox vaccinations increases with the prevalence of contagious diseases.

By making the distinction between the intensive and the extensive margins of colonial investments, the next section will allow to describe more precisely the provision's strategy for colonial policies.

### 1.6.2 Intensive and extensive margins of colonial investments

Table 1.3 shows estimation results of the Cragg's model, where $y$ in Equation (1.2) is the number of African medical staff. Table 1.4 shows results of the Cragg's model, where $y$ is the number of smallpox vaccinations. Last, Table 1.6 shows results for the number of teachers. Comments focus on $x$'s for which $\beta/\sigma$ is different from $\gamma$ in Equation (1.2), i.e., determinants of colonial inputs whose estimated coefficients for the first and the second tier are not equal.

**Table 1.3:** African medical staff's determinants - Cragg's model

| | African Medical Staff | | |
|---|---|---|---|
| | First tier $\frac{\partial P(y>0\|x_1)}{\partial x_j}$ | Second tier $\frac{\partial \mathbb{E}(y\|y>0,x_2)}{\partial x_j}$ | APE $\frac{\partial \mathbb{E}(y\|x_1,x_2)}{\partial x_j}$ |
| **The structure of the colonial system:** | | | |
| $\mathbb{1}${Medical center in $t-1$>0} | 0.535*** | 4.965* | 7.835*** |
| | (0.128) | (2.446) | (1.897) |
| Colonial staff in $t-1$ | -0.002 | 0.039** | 0.010 |
| | (0.001) | (0.016) | (0.014) |
| Strong presence of health adm. in $t-1$ | 0.127 | 0.581 | 1.490 |
| | (0.092) | (1.142) | (1.059) |
| Strong presence of other adm. in $t-1$ | -0.313** | -1.852 | -3.932** |
| | (0.127) | (1.727) | (1.542) |
| **Context:** | | | |
| $\mathbb{1}${Cities in $t-1$} | 0.107 | 3.698** | 3.227*** |
| | (0.077) | (1.373) | (1.074) |
| **Diseases:** | | | |
| $\mathbb{1}${Contagious diseases in $t-1$>0} – low prevalence | 0.154* | -1.755 | 0.299 |
| | (0.090) | (1.076) | (1.036) |
| $\mathbb{1}${Contagious diseases in $t-1$>0} – high prevalence | 0.235** | -2.279** | 0.702 |
| | (0.098) | (1.062) | (1.087) |
| $\mathbb{1}${Non-contagious diseases in $t-1$>0} | 0.294*** | 0.511 | 2.937*** |
| | (0.089) | (0.674) | (0.894) |
| Observations | 697 | 697 | 697 |
| Colony and Period F.E. | Yes | | |
| District and Period F.E. | | Yes | |
| Mean investment | 0.65 | 19.12 | 12.42 |

See Table 1.1 for notes. Full estimation of Cragg's model for African Medical staff. Average partial effects and standard errors (in parentheses) are obtained with the delta method. Mean investment: $P(y>0)$ (first column), $\mathbb{E}(y|y>0)$ (second column) and $\mathbb{E}(y)$ (third column).

**Table 1.4:** Vaccinations' determinants - Cragg's model

| | Vaccinations | | |
|---|---|---|---|
| | First tier $\frac{\partial P(y>0\|x_1)}{\partial x_j}$ | Second tier $\frac{\partial \mathbb{E}(y\|y>0,x_2)}{\partial x_j}$ | APE $\frac{\partial \mathbb{E}(y\|x_1,x_2)}{\partial x_j}$ |
| **The structure of the colonial system:** | | | |
| $\mathbb{1}${Medical center in $t-1$>0} | 0.186*** | -5.390 | 14.473* |
| | (0.039) | (8.952) | (8.424) |
| Colonial staff in $t-1$ | -0.001 | -0.034 | -0.097 |
| | (0.001) | (0.119) | (0.125) |
| Strong presence of health adm. in $t-1$ | 0.123*** | -19.898 | -4.315 |
| | (0.045) | (12.009) | (11.177) |
| Strong presence of other adm. in $t-1$ | 0.037 | -4.606 | -0.111 |
| | (0.057) | (11.059) | (10.863) |
| **Context:** | | | |
| $\mathbb{1}${Cities in $t-1$} | 0.168*** | 7.368 | 23.452** |
| | (0.054) | (9.291) | (9.776) |
| **Diseases:** | | | |
| $\mathbb{1}${Contagious diseases in $t-1$>0} – low prevalence | 0.062 | 10.404 | 15.123* |
| | (0.047) | (9.211) | (9.050) |
| $\mathbb{1}${Contagious diseases in $t-1$>0} – high prevalence | 0.058 | 18.758 | 21.902* |
| | (0.056) | (11.377) | (11.170) |
| $\mathbb{1}${Non-contagious diseases in $t-1$>0} | 0.108*** | 1.479 | 12.325 |
| | (0.045) | (10.736) | (10.344) |
| Observations | 952 | 952 | 952 |
| Colony and Period F.E. | Yes | | |
| District and Period F.E. | | Yes | |
| Mean investment | 0.8 | 151.62 | 121.8 |

See Tables 1.1 and 1.3 for notes. Full estimation of Cragg's model for vaccinations. Average partial effects and standard errors (in parentheses) are obtained with the delta method.

### The specificity of health investments: "coverage" and "diversification"

The basic questions asked here are whether already provisioned districts get more or less colonial investments than others, and what are the underlying mechanisms.

The presence of a medical center in the previous period has a positive and strong impact on the probability to place an health input in district $z$ and period $t$. The probability to find African medical staff in the district is 54% higher if there was a medical center. Impacts on vaccinations' provision are smaller but significant at the one percent level. The probability to implement smallpox immunization is 18% higher if a medical center existed. The impact of this infrastructure's presence on the *quantity* of health inputs is either non significant – vaccinations – either much smaller – African medical staff. Then, both health investments also correlate to a strong presence of health services in $z$ and $G(z)$: the probability to find vaccinations or medical staff increases by around $12-13\%$ if there was a strong presence of health administrations (only significant for vaccinations). This pattern of the colonial system does not matter for the quantity of investments. Under the plausible assumption that returns to scale are higher within the same type of investments, these findings point towards a high fixed cost of health investments leading to the targeting of districts that are not medical deserts.

This fixed cost channel also matters for other investments, and is not specific to health inputs. However, the strategy of the colonial administration regarding health investments is specific regarding two dimensions.

First, the provision of both vaccinations and medical staff results from a "diversification" strategy. This conclusion derives from the correlation of colonial investments with lagged investments on a sub-sample.[27] Table 1.5 estimates a linear dynamic panel-data model, including the lag of the dependent variable as a further covariate. The estimated equation is:

$$I_{z,t} = \alpha_1 + \alpha_2.I_{z,t-1} + \beta.System_{z,t-1} + \gamma.Context_{z,t-1} + \delta.Diseases_{z,t-1} + \lambda_t + \mu_z + \epsilon_{zt} \quad (1.3)$$

A consistent generalized method of moments estimator is obtained for the parameters of this model, using the method derived by Arellano and Bond (1991). Health investments are negatively correlated to lagged investments: five more staff in $t-1$ lead to around one less staff in $t$; six more lagged vaccinations lead to around one less vaccination in $t$. This finding could be interpreted as a long-lasting impact of investments: districts who got more medical care in one period are already better protected in the following period. [28]

**Table 1.5:** Dynamic panel-data model: the link to the lagged stock of investments

| | Colonial Investments ($y$) | | | |
|---|---|---|---|---|
| | **African Med. Staff** per 100,000 hab. | **Smallpox Vaccinations** per 1,000 hab. | **Teachers** | **Nb Conscripts** per hab. |
| Lagged $y$ | -0.226* | -0.166** | 0.309*** | 0.041 |
| | (0.129) | (0.068) | (0.072) | (0.029) |
| Observations | 175 | 313 | 322 | 1259 |
| District and Period F.E. | Yes | Yes | Yes | Yes |

*** p<0.01, ** p<0.05, * p<0.1. The estimation of lagged $y$ is obtained with the estimation method of Arellano and Bond (1991). All variables of Table 1.1 are also included in the estimation. The sample size is reduced compared to other tables because of the estimation method (see text for details).

Second, as already implied by linear results, the strategy of allocation for medical staff is rather complex. On the one hand, there is a positive correlation of trends between medical staff and colonial staff, which is driven by the quantity of inputs (second tier). On the other hand, the probability to find medical staff in a given district is more than 30% lower if there is a strong presence of other administrations. All in all, the allocation of medical staff follows a two-level strategy: *(i)* medical staff are used as a means of general "coverage" – one incentive to elect a district to receive medical staff is the fact that the colonial administration is not much developed yet; *(ii)* then, some already provisioned districts benefit from path dependence or returns to scale, and get more staff.

Compared to those findings, the strategy for other colonial investments differs a lot. Both the probability to find teachers in a district and the quantity of teachers increase with the pres-

[27] Given the method of estimation used, the sample size is reduced to observations for which first and second order lags are not missing for both the endogenous and the explanatory variables. Public Works' results are not shown in this table: they were computed on a very small sample, because there are important time gaps in the data.

[28] Similarly, during the beginning of the colonial period, the amount of public works' expenses decreases with the colonial presence: 40% to 50% less are spent if there is a strong presence of health or other administrations (Table A-1.5). This finding can also be reconciled with a long-lasting impact of public works' investments.

ence of colonial administration (Table 1.6). The fact that the colonial system matters in both tiers provides evidence that teachers' allocation depends on cost minimization, but that it is also guided by a path dependence strategy. However, contrary to medical staff, teachers are not allocated following a two-level strategy, by which "diversification" or "coverage" would also matter. This last point is confirmed by the fact that educational investments are positively correlated with lagged investments: three more teachers in $t-1$ lead to around one more teacher in $t$ (Table 1.5).

**Table 1.6:** Teachers' determinants - Cragg's model

| | Teachers | | |
|---|---|---|---|
| | First tier $\frac{\partial P(y>0\|x_1)}{\partial x_j}$ | Second tier $\frac{\partial \mathbb{E}(y\|y>0,x_2)}{\partial x_j}$ | APE $\frac{\partial \mathbb{E}(y\|x_1,x_2)}{\partial x_j}$ |
| **The structure of the colonial system:** | | | |
| $\mathbb{1}${Medical center in $t-1$>0} | 0.019 | -0.057 | 0.052 |
| | (0.032) | (0.505) | (0.523) |
| Colonial staff in $t-1$ | -0.001 | 0.039 | 0.033 |
| | (0.001) | (0.027) | (0.025) |
| Strong presence of health adm. in $t-1$ | 0.052** | 1.897*** | 2.080*** |
| | (0.025) | (0.621) | (0.602) |
| Strong presence of other adm. in $t-1$ | 0.059** | 0.465 | 0.771 |
| | (0.025) | (0.594) | (0.576) |
| **Context:** | | | |
| $\mathbb{1}${Cities in $t-1$} | 0.095*** | 2.980*** | 3.345*** |
| | (0.031) | (0.703) | (0.681) |
| **Diseases:** | | | |
| $\mathbb{1}${Contagious diseases in $t-1$>0} – low prevalence | -0.038 | 1.594*** | 1.283** |
| | (0.034) | (0.587) | (0.590) |
| $\mathbb{1}${Contagious diseases in $t-1$>0} – high prevalence | 0.036 | 0.699 | 0.860 |
| | (0.036) | (0.669) | (0.674) |
| $\mathbb{1}${Non-contagious diseases in $t-1$>0} | 0.056* | -1.034 | -0.655 |
| | (0.032) | (0.690) | (0.664) |
| Observations | 662 | 662 | 662 |
| Colony and Period F.E. | Yes | | |
| District and Period F.E. | | Yes | |
| Mean investment | 0.87 | 6.18 | 5.40 |

See Tables 1.1 and 1.3 for notes. Full estimation of Cragg's model for teachers. Average partial effects and standard errors (in parentheses) are obtained with the delta method.

The allocation of staff – both medical and educational – is guided by a path dependence strategy. This strategy can be described as a general – rather than specific – principle of investments' complementarity. There was no targeted specialization of investments at the district level. Regarding education, in Table 1.6, the quantity of teachers increases more if there is a strong presence of health administration than if there is a strong presence of other administrations. Regarding health, the quantity of medical staff decreases with the lagged stock of staff (Table 1.5).

All in all, the dynamic strategy for the allocation of health inputs and for the allocation of teachers are not the same. All inputs are allocated in priority to places where the cost is smaller through returns to scale. Consequently, the allocation of medical staff and vaccinations correlates positively to some dimensions of the past colonial system. However, if the allocation

of teachers is driven by path dependence and returns to scale only, it is not the case for health inputs. For medical staff, there is also a strategy to "cover" isolated districts. Moreover, districts who got more vaccinations or medical staff in one period tend to have less in the following period, through the long-lasting effect of prevention.

**The lower cost of all colonial investments in cities**

For African medical staff and teachers, the presence of cities mainly matters for intensive investments' decisions. It does not matter, or matters much less for the probability that colonial staff is found in the district. On the contrary, urban presence increases the probability to vaccinate by 17 percentage points, and has no impact on the number of vaccinations. These findings can easily be reconciled with a scenario of increasing returns to population for investments. Indeed, it is very likely that it is less costly to vaccinate in densely populated areas, but that conditionally on vaccinations taking place, the cost per vaccine is not related to the population context. On the contrary, one can argue that it is less costly to increase the number of colonial staff in urban areas, even conditionally on staff being provisioned in the district.

It would be much harder to reconcile the empirical findings with a story of an intrinsic colonial motivation to favor urban or European populations. In this second less likely scenario, there is no reason why preferences would be stronger during the beginning of the colonial period, and no reason why preferences would not change according to the type of colonial investments.

**Disease prevalence proxies the demand and the contagion risk**

The prevalence of non-contagious diseases is a significant predictor of all colonial inputs studied in this section (medical and educational staff, and vaccinations) in the first tier only. The reporting of a positive number of casualties from non-contagious diseases in the previous period increases the probability to provision African medical staff by 29 percentage points, the probability to provision teachers by eleven percentage points, and the probability to vaccinate by six percentage points. Together with the fact that colonial investments other than health inputs increase with the prevalence of non-contagious diseases, these findings provide suggestive evidence that the prevalence of non-contagious diseases is related to colonial inputs through demand. Colonial investments targeted places in which the demand for colonial policies was greater.

The probability to find medical staff is also 15 to 23% higher if contagious diseases were prevalent in the district; which is mainly driven by the end of the colonial period. Given that the quantity of medical staff decreases with contagious prevalence, the underlying mechanism is most probably an answer of the administration to a greater demand for health. The preva-

lence of contagious diseases matters for the allocation of smallpox vaccinations, in particular in the end of the colonial period. There is no significant difference between the first and the second tier equations regarding this determinant. This finding confirms that the allocation of vaccinations was guided by districts' contagion risk. In any case, given that most vaccinations' campaigns were compulsory under the colonial rule, the demand or "sympathy" channel must be excluded for this outcome. Last, the contagion risk matters for the quantity of teachers allocated to a given district; this result is driven by the beginning of the colonial period. This last finding is in line with the qualitative evidence found in health reports according to which schools were used to provide diseases' prevention.

### 1.6.3 Robustness checks

Results are robust to removing weights, thus giving the same weight to each district×period. The main difference when removing weights is that results are stronger and more significant for the impact of contagious diseases, indicating that these effects are mostly driven by less populated districts. Weighting the regressions and giving less weight to these districts tends to mitigate these results.

Results of Section 1.6.2 are robust to a bootstrapping procedure. Bootstrapping starts by reestimating the model and generating a new Average Partial Effect (APE) on a random subsample within the data. This process is iterated 200 times until many APEs have been computed from numerous random sub-samples. The standard deviations from those APEs are then used as a standard error for the full sample average partial effect. Bootstrap estimation results are available on request.

## 1.7 Conclusion

Colonial data show a big increase in colonial investments over the period 1904 to 1958 – health, education, public works and military expenses. The positive trend observed for health provision is driven by a greater increase in health care provided by African staff and nurses, outside of hospitals. Health inputs' maps display a big heterogeneity of health provision across districts. More generally, there is a great heterogeneity of colonial services' provision across districts: early urbanized districts get proportionally more investments, and a high concentration of inputs' provision towards Europeans is observed. Such heterogeneity decreases over time, apart from the factors related to hospital-related health care and military expenses.

This paper describes the decision-making process of health provision within the colonial system. It draws potential scenarii describing the allocation of colonial investments, and questions whether health investments' decisions were generic or specific. More precisely, three main questions are asked regarding the allocation's strategy of colonial policies. First, how do

colonial investments relate to the existing colonial system? What is the dynamic strategy of investments? Second, does the population context matter for the allocation of colonial policies? Are some specific populations targeted, all things being equal? Third, what is the impact of disease prevalence on the provision of colonial services? Does disease prevalence capture health needs and contagion risk only, or the demand for colonial services as well?

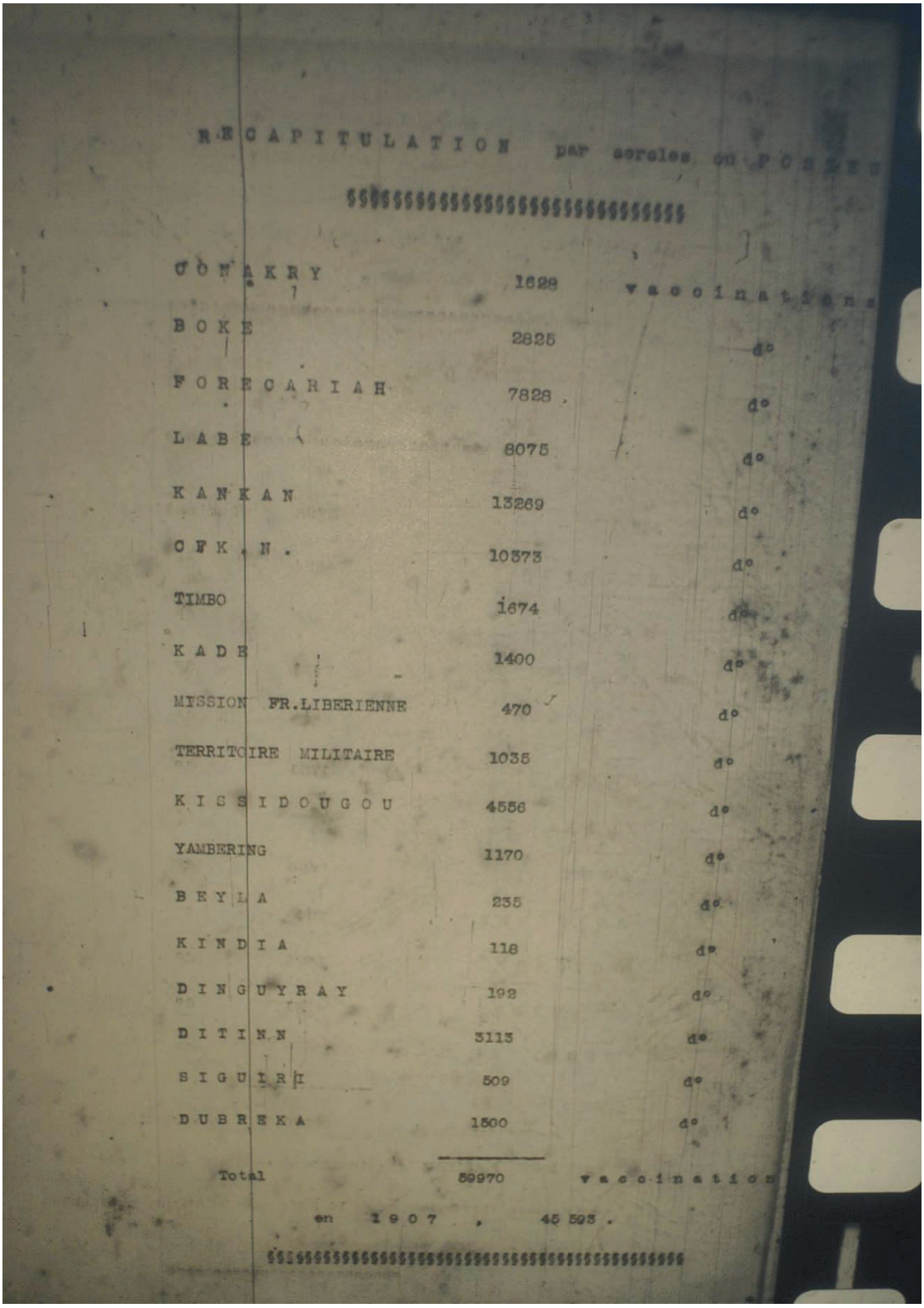
This work eventually demonstrates that there exists a very general strategy regarding the provision of colonial services. Investments take place in priority in districts where returns to scale are higher, and where investments are thus less costly. Moreover, all colonial inputs increase with the proportion of urban population, the proportion of Europeans, or the population density. This result is explained by increasing returns to population, which are driving down the cost of investment in densely populated areas. This paper provides further evidence that the prevalence of non-contagious diseases is related to colonial inputs through demand. All things being equal, all colonial investments target places in which the demand for colonial policies, as proxied by the prevalence of non-contagious diseases, is greater. Colonial investments also correlate to contagion risk; the most robust results being found for smallpox vaccinations. Last, the allocation for both medical and educational staff is guided by a general – rather than specific – principle of investments' complementarity. There was no targeted specialization of investments at the district level.

The main distinction between health inputs and other inputs such as education relates to what can be described as the dynamic strategy of investments. The allocation of teachers is self-reinforcing; and is driven by path dependence and returns to scale. If health investments also correlate positively to some dimensions of the past colonial system, their provision is specific in two dimensions. First, medical staff is used as a means of colonial "coverage". Second, all health investments follow a "diversification" strategy related to the long-lasting effect of prevention, which does not exist for education.

Colonial health policies implemented in former French West Africa were far from being exogenous. A detailed analysis of the colonial health system shows that the development of health policies took place in close relation with the broader development of all colonial policies. Further research is needed to reconcile the end of the colonial period with the post-colonial period. What happened at colonial independence? Did colonial health inputs' trends persist? What was the post-colonial strategy for the provision of public investments?

# Appendix

**Figure A-1.1:** Example of archive extract: vaccinations in 1922 Guinea



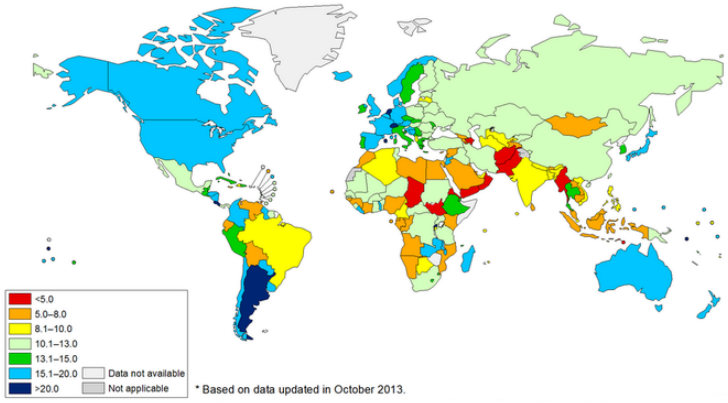
RECAPITULATION par cercles ou POSTES

| | | |
|---|---|---|
| CONAKRY | 1628 | vaccinations |
| BOKE | 2825 | d° |
| FORECARIAH | 7828 | d° |
| LABE | 8075 | d° |
| KANKAN | 13269 | d° |
| CFK. N. | 10373 | d° |
| TIMBO | 1674 | d° |
| KADE | 1400 | d° |
| MISSION FR.LIBERIENNE | 470 | d° |
| TERRITOIRE MILITAIRE | 1035 | d° |
| KISSIDOUGOU | 4556 | d° |
| YAMBERING | 1170 | d° |
| BEYLA | 235 | d° |
| KINDIA | 118 | d° |
| DINGUYRAY | 198 | d° |
| DITINN | 3113 | d° |
| SIGUIRI | 509 | d° |
| DUBREKA | 1500 | d° |
| Total | 59970 | vaccination |

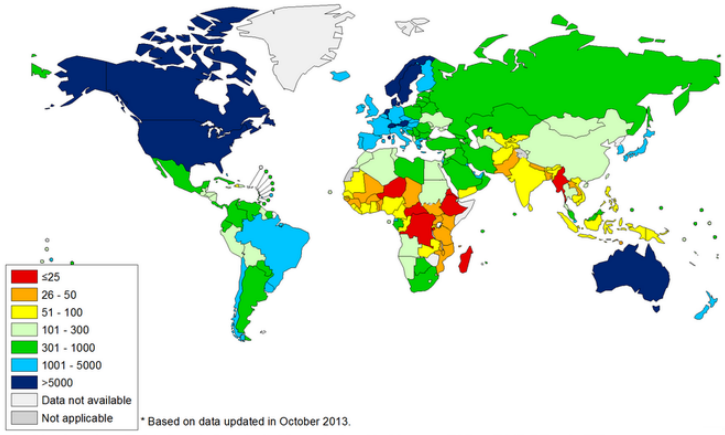
en 1907 , 45 593 .

**Figure A-1.2:** Health expenditures in the world, 2013

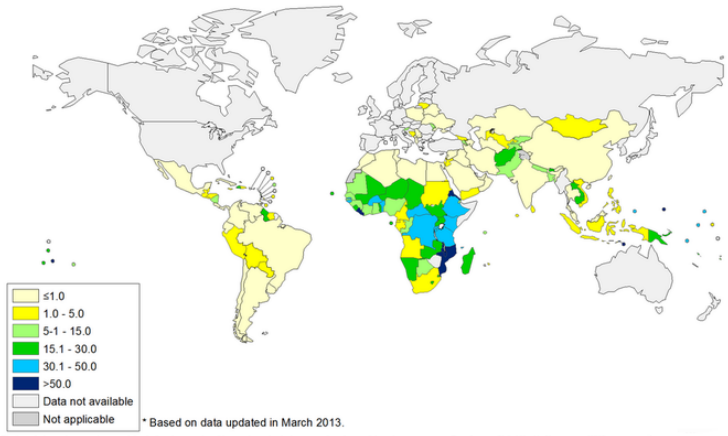
Government expenditures on health (% of total gov. exp.)




Per capita total health expenditures, at average exchange rate (USD), 2011




External resources (% of total health exp.)




Source: World Health Organization, 2013.

**Figure A-1.3:** Maps of former French West Africa: medical staff provision

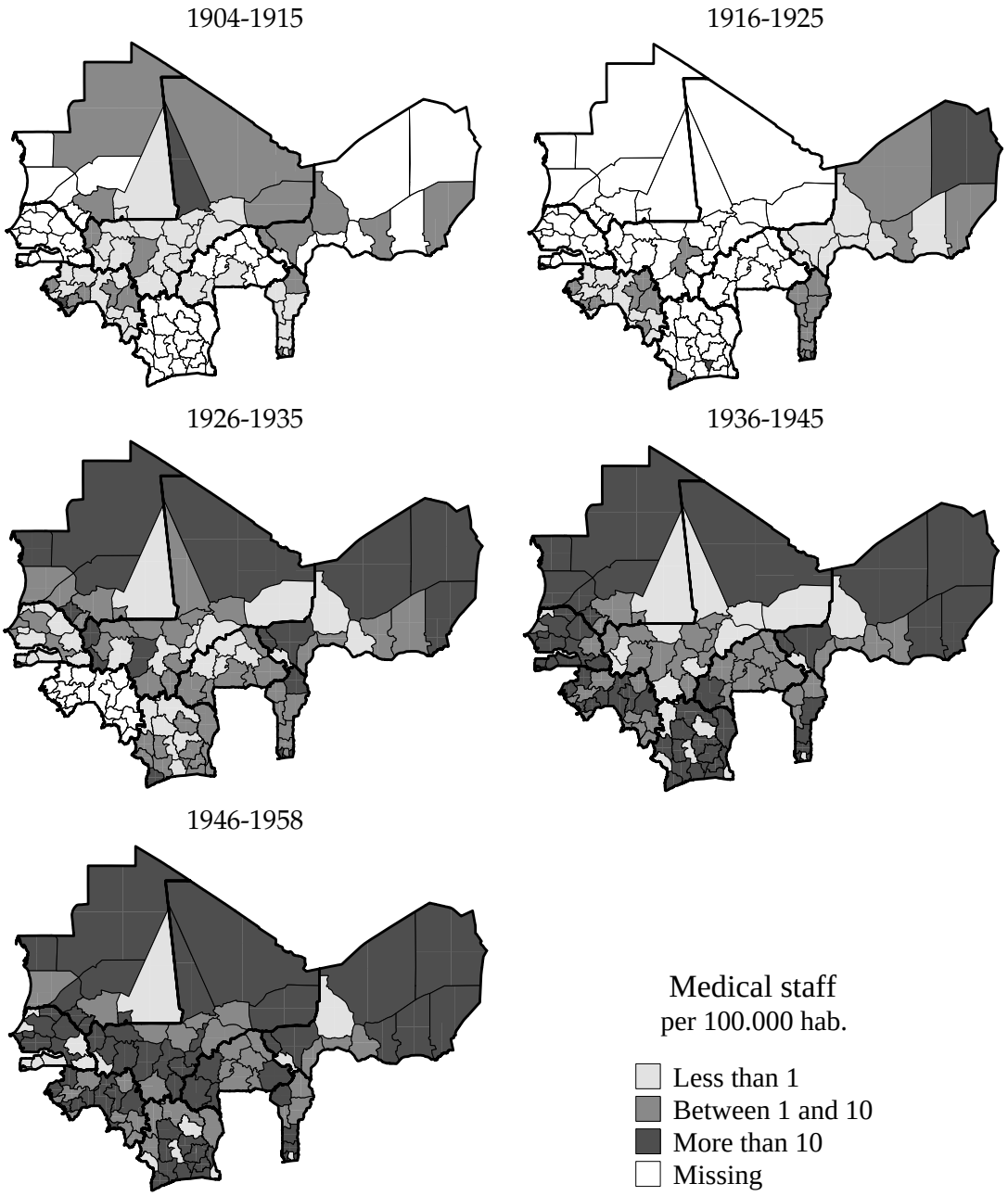


**Table A-1.1:** Colonial health policies in former French West Africa

| Year | Health Policy | Dahomey | Guinea | Ivory Coast | Mauritania |
|---|---|---|---|---|---|
| 1904-1915 | **Treated diseases** | Leprosy, malaria, syph., tuber. | Leprosy, malaria, syph., tuber., tryp. | Leprosy, malaria, syph., tuber. | " |
| | **Surveyed diseases** | Beriberi, bilious f., filar., mumps | Beriberi, bilious f., chick., filar., leprosy, malaria, measles, mumps, smallpox, syph., tryp., tuber., w. cough | Beriberi, bilious f., chick., filar., mumps, smallpox, tetanus, tryp., yellow f. | N/A |
| | **Surveyed deaths** | N/A | Smallpox, tryp., tuber. | Smallpox | N/A |
| | **Vaccinations** | Smallpox | " | " | " |
| | **Other** | N/A | Ass. deliveries for Europeans | N/A | N/A |
| 1916-1925 | **Treated diseases** | N/A | Malaria | Leprosy, malaria, mening., rabies, syph., tuber. | N/A |
| | **Surveyed diseases** | Bilious f., chick., filar., leprosy, malaria, mumps, smallpox, syph., tuber. | Bilious f., chick., dysentery, filar., leprosy, measles, mumps, smallpox, syph, tryp., tuber., yaws | Beriberi, chick., dysentery, filar., measles, pneum., smallpox, syph., tryp. | Mening., plague, smallpox |
| | **Surveyed deaths** | N/A | N/A | Leprosy, malaria, smallpox, syph., tryp., tuber. | Mening., plague, smallpox |
| | **Vaccinations** | Smallpox | " | Smallpox, yellow f. | Smallpox |
| | **Other** | N/A | N/A | Ass. deliveries | N/A |
| 1926-1935 | **Treated diseases** | Malaria, tuber. | Leprosy, malaria | Malaria, tuber. | N/A |
| | **Surveyed diseases** | Chick., dysentery, leprosy, measles, mumps, pneum., smallpox, trachoma, tryp., tuber., yaws | Chick., dysentery, measles, mumps, smallpox, w. cough, yellow f. | Bilious f., chick., dysentery, filar., leprosy, measles, mumps, pneum., smallpox, trachoma, tryp., tuber., yaws, yellow f. | Leprosy, smallpox |
| | **Surveyed deaths** | Pneum., smallpox, tryp., yellow f. | Smallpox, yellow f. | Pneum., smallpox, tryp., yellow f. | N/A |
| | **Vaccinations** | Smallpox, yellow f. | Smallpox | Smallpox, yellow f. | Smallpox |
| | **Other** | Ass. deliveries, pre/postnatal & inf. consult. | " | " | N/A |
| 1936-1945 | **Treated diseases** | Leprosy, tryp. | " | " | Leprosy |
| | **Surveyed diseases** | Leprosy, mening., smallpox, tetanus, yellow f. | Leprosy, mening., smallpox, tryp., ulcer | Leprosy, mening., smallpox, tetanus, yellow f. | Leprosy, malaria, mening., smallpox |
| | **Surveyed deaths** | Mening., smallpox, tryp., yellow f. | Leprosy, mening., smallpox, tryp. | Smallpox, tryp. | Mening., malaria, pneum., smallpox, tetanus, tuber., yellow f. |
| | **Vaccinations** | Mening., smallpox, yellow f. | Smallpox, yellow f. | Mening., smallpox, yellow f. | Smallpox |
| | **Other** | N/A | Ass. deliveries | Abortion, ass. deliveries, prenatal & inf. consult. | N/A |
| 1946-1958 | **Treated diseases** | Leprosy | Leprosy, tryp. | Leprosy | " |
| | **Surveyed diseases** | Bilious f., leprosy, malaria, mening., smallpox, typhoid, tuber. | Leprosy, mening., plague, polio, smallpox, tryp., w. cough | Bilious f., leprosy, malaria, mening., smallpox, typhoid, tuber. | Leprosy |
| | **Surveyed deaths** | Leprosy, smallpox | Leprosy, mening., plague, smallpox, tryp. | Leprosy, smallpox | Leprosy |
| | **Vaccinations** | Smallpox, tuberculosis | Cholera, plague, smallpox, tuberculosis, yellow f. | Smallpox, tuberculosis | Smallpox, tuberculosis, yellow f. |
| | **Other** | Ass. deliveries, pre/postnatal consult., school prevention | School prevention | Abortion, ass. deliveries, pre/postnatal & inf. consult., school prevention | Abortion, ass. deliveries, pre/postnatal consult. |

Data extracted from the colonial administrative health reports of former French West Africa. Ass. deliveries: assisted deliveries; bilious f.: bilious fever; chick.: chickenpox; consult.: consultations; filar.: filariasis; inf.: infant; mening.: meningitis; pneum.: pneumonia; syph.: syphilis; tryp: trypanosomiasis; tuber.: tuberculosis; w.cough : whooping cough; yellow f.: yellow fever

| Year | Health Policy | Niger | Senegal | Soudan | Upper Volta |
|---|---|---|---|---|---|
| 1904- | **Treated diseases** | Malaria, syph., tuber. | Leprosy, malaria, syph., tryp., tuber. | Malaria, mening., syph., tryp., tuber. | Malaria, syph., tryp., tuber. |
| 1915 | **Surveyed diseases** | Bilious f., dysentery, filar., measles, smallpox, syph. | Beriberi, bilious f., filar., measles, plague, smallpox, tryp., tuber. | Beriberi, chick., dysentery, filar., leprosy, measles, mening., mumps, smallpox, syph., tryp., tuber. | Dysentery, filar., mumps, smallpox, syph., tryp., tuber., w. cough |
| | **Surveyed deaths** | N/A | Plague | N/A | N/A |
| | **Vaccinations** | Smallpox | " | " | " |
| | **Other** | N/A | N/A | N/A | N/A |
| 1916- | **Treated diseases** | Leprosy, malaria, syph., tuber. | N/A | N/A | N/A |
| 1925 | **Surveyed diseases** | Beriberi, bilious f., filar., leprosy, malaria, syph., tryp., tuber. | Plague | Measles, mening., periodic f., smallpox, tryp., typhoid | Periodic f. |
| | **Surveyed deaths** | Syph., tuber. | Plague | N/A | Periodic f. |
| | **Vaccinations** | Smallpox | Smallpox, tuberculosis | Smallpox | N/A |
| | **Other** | N/A | Ass. deliveries | " | N/A |
| 1926- | **Treated diseases** | N/A | Leprosy, malaria, mening, syph., tuber. | N/A | Tryp. |
| 1935 | **Surveyed diseases** | Bilharzia, chick., filar., measles, mumps, smallpox, tryp., w.cough, yellow f. | Bilharzia, bilious f., chick., diphtery, dysentery, gonorrhoea, malaria, measles, mening., mumps, plague, smallpox, tetanus, tryp., ulcer, yellow f. | Chick., dysentery, measles, mening., mumps, periodic f., plague, smallpox, typhoid, tryp., w. cough, yellow f. | Bilharzia, chick., filar., measles, mumps, periodic f., smallpox, tryp., w. cough, yellow f. |
| | **Surveyed deaths** | Periodic f., smallpox, tryp., yellow f. | Malaria, plague, smallpox, tuber., yellow f. | Mening., periodic f., plague, smallpox, yellow f. | Periodic f., tryp., yellow f. |
| | **Vaccinations** | Smallpox | Diphteria, mening., plague, smallpox, tuberculosis yellow f. | " | Smallpox, yellow f. |
| | **Other** | Ass. deliveries, inf. consult. | Abortion, ass. deliveries, prenatal & infant consult. | Ass. deliveries, prenatal & inf. consult. | " |
| 1936- | **Treated diseases** | Leprosy, malaria, mening., tryp. | Amoebiasis, assisted deliveries, prenatal/infant consultations leprosy, malaria, rabies, syph., tryp., tuber. | Leprosy, rabies, tryp. | Leprosy, malaria, tryp. |
| 1945 | **Surveyed diseases** | Bilharzia, chancroid, filar., gonorrhoea, leprosy, malaria, measles, mening., mumps, pneum., smallpox, syph., trachoma, tuber., yaws, yellow f. | Beriberi, bilharzia, bilious f., chancroid, chick., diphtheria, dysentery, filar., leprosy, gonorrhoea, malaria, measles, mening., mumps, periodic f., plague, pneum., smallpox, syph., tetanus, trachoma, tuber., typhoid, ulcer, w. cough, yaws, yellow f. | Leprosy, mening., smallpox, tetanus, trachoma, tryp., yellow f. | Bilharzia, chancroid, gonorrhoea, measles, mumps, filar., leprosy, malaria, measles, mening., mumps, pneum., smallpox, syph., tetanus, trachoma, tryp., tuber., yaws, yellow f. |
| | **Surveyed deaths** | Malaria, mening., pneum., smallpox., tuber., yellow f. | Leprosy, malaria, mening., periodic f., plague, smallpox, tetanus, tuber., yellow f. | Mening., smallpox, tetanus, yellow f. | Leprosy, malaria, mening., pneum., smallpox, tuber. |
| | **Vaccinations** | Mening., smallpox, yellow f. | Cholera, diphtheria, mening., plague, pneum., rabies, smallpox, tuberculosis yellow f. | Mening., smallpox, yellow f. | Smallpox, yellow f. |
| | **Other** | Abortion, ass. deliveries, pre/postnatal & inf. consult. | " | " | " |
| 1946- | **Treated diseases** | Leprosy | Leprosy, syph., tuber. | Leprosy, tryp. | " |
| 1958 | **Surveyed diseases** | Leprosy, mening., smallpox, syph., tuber. | Bilharzia, chancroid, chick., diphtery, dysentery, leprosy, malaria, measles, mening., mumps, periodic f., polio, smallpox, syph., tetanus, trachoma, tuber., typhoid, ulcer, w.cough, yaws | Beriberi, bilious f., chancroid, chick., diphtheria, leprosy, malaria, measles, mening., mumps, periodic f., smallpox, syph., tetanus, trachoma, tryp., tuber., typhoid, w.cough, yaws | Bilharzia, diphtheria, dysentery, leprosy, malaria, measles, mening., mumps, periodic f., pneum., smallpox, syph., trachoma, tryp., tuber., typhoid, w. cough, yaws |
| | **Surveyed deaths** | Leprosy, smallpox | Leprosy, mening., syph., tetanus, tryp., tuber. | Leprosy, malaria, mening., syph., smallpox, tetanus, tryp., tuber. | Leprosy, malaria, mening., pneum., smallpox, tuber. |
| | **Vaccinations** | Diphtheria, smallpox, tuberculosis yellow f. | Cholera, plague, smallpox, tuberculosis, yellow f. | Plague, pneum., rabies, smallpox, tuberculosis | Tuberculosis |
| | **Other** | Ass. deliveries, school prevention | Abortion, ass. deliveries, pre/postnatal & inf. consult., school prevention | Abortion, ass. deliveries, pre/postnatal & inf. consult. | Ass. deliveries, pre/postnatal & inf. consult., school prevention |

**Table A-1.2:** Colonial budget allocated to health, by district

| Colony | District | Year | Budget (Fcs) | Population |
|---|---|---|---|---|
| Upper-Volta (Burkina Faso) | Dedougou | 1936 | 25,000 | 387,500 |
| Upper-Volta (Burkina Faso) | Gaoua | 1936 | 73,000 | 230,800 |
| Upper-Volta (Burkina Faso) | Kaya | 1936 | 25,000 | 264,900 |
| Upper-Volta (Burkina Faso) | Koudougou | 1936 | 5,000 | 349,600 |
| Upper-Volta (Burkina Faso) | Ouagadougou | 1936 | 12,7500 | 470,300 |
| Upper-Volta (Burkina Faso) | Tenkodogo | 1936 | 25,000 | 204,800 |
| Cote d'Ivoire | Baoule | 1936 | 1,400 | 255,700 |
| Cote d'Ivoire | Daloa | 1936 | 25,000 | 92,300 |
| Cote d'Ivoire | Guiglo | 1936 | 10,000 | 36,200 |
| Cote d'Ivoire | Lahou | 1936 | 15,000 | 80,300 |
| Cote d'Ivoire | Man | 1936 | 25,000 | 218,200 |
| Cote d'Ivoire | Odienne | 1936 | 15,000 | 131,100 |
| Cote d'Ivoire | Sassandra | 1936 | 30,000 | 93,600 |
| Cote d'Ivoire | Seguela | 1936 | 25,000 | 103,300 |
| Cote d'Ivoire | Tabou | 1936 | 25,000 | 17,200 |
| Cote d'Ivoire | Tagouanas | 1936 | 400 | 62,800 |
| Cote d'Ivoire | Baoule | 1958 | 4,201,000 | 327,600 |
| Cote d'Ivoire | Bassam | 1958 | 746,000 | 40,800 |
| Cote d'Ivoire | Daloa | 1958 | 200,000 | 108,800 |
| Cote d'Ivoire | Lagunes | 1958 | 8,003,000 | 189,400 |
| Cote d'Ivoire | Nzi Comoe | 1958 | 1,789,000 | 190,400 |
| Cote d'Ivoire | Sassandra | 1958 | 1,615,613 | 128,300 |

These very incomplete data on health colonial budget are extracted from colonial archives.

**Table A-1.3:** Descriptive statistics: baseline (1904-1915) and mean values

| | Mean | SD |
|---|---|---|
| *Per 100,000 hab.* | | |
| Physicians: | | |
| - baseline stock | 1.10 | 0.86 |
| - mean stock | 2.72 | 2.54 |
| Nurses: | | |
| - baseline stock | 0.40 | 0.58 |
| - mean stock | 8.93 | 11.61 |
| African Staff: | | |
| - baseline stock | 0.33 | 0.65 |
| - mean stock | 12.61 | 17.64 |
| Medical Staff: | | |
| - baseline stock | 1.62 | 0.77 |
| - mean stock | 16.55 | 18.96 |
| *Per 1,000 hab.* | | |
| Smallpox Vaccinations: | | |
| - baseline stock | 42.51 | 21.95 |
| - mean stock | 133.11 | 132.65 |
| Consultants: | | |
| - baseline stock | 28.74 | 33.46 |
| - mean stock | 209.99 | 401.41 |
| Consultations: | | |
| - baseline stock | 99.79 | 105.93 |
| - mean stock | 617.15 | 1,268.32 |
| Hospital days: | | |
| - baseline stock | 118.14 | 175.72 |
| - mean stock | 421.48 | 1,013.56 |
| Nb of Europeans: | | |
| - baseline stock | 1.23 | 1.54 |
| - mean stock | 4.31 | 7.78 |
| Nb of Urbans: | | |
| - baseline stock | 17.21 | 36.37 |
| - mean stock | 58.78 | 106.24 |

All staff variables are expressed as per $100,000$ 1925 capita. Other health variables are expressed as per $1,000$ 1925 capita.

**Table A-1.4:** Health inputs' determinants - across two periods

| | All African staff per 100,000 hab. | | Vaccinations per 1,000 hab. | |
|---|---|---|---|---|
| | **1908-42** | **1943-58** | **1908-31** | **1932-58** |
| **The structure of the colonial system:** | | | | |
| $\mathbb{1}$\{Medical center in $t-1$>0\} | 2.589 | 20.533** | 28.688 | 167.193** |
| | (2.152) | (10.229) | (20.244) | (76.909) |
| Colonial staff in $t-1$ | -0.041 | 0.337** | 1.931** | -0.091 |
| | (0.128) | (0.163) | (0.934) | (0.648) |
| Strong presence of health adm. in $t-1$ | 0.281 | 4.452 | 3.954 | -232.971 |
| | (2.703) | (5.518) | (14.705) | (208.025) |
| Strong presence of other adm. in $t-1$ | -5.075 | -9.957 | 7.166 | -45.828 |
| | (3.268) | (15.934) | (27.779) | (80.739) |
| **Context:** | | | | |
| $\mathbb{1}$ {Cities in $t-1$} | 6.701** | 10.738* | 68.721 | 34.143 |
| | (3.274) | (5.532) | (50.493) | (45.783) |
| **Diseases:** | | | | |
| $\mathbb{1}$\{Contagious diseases in $t-1$>0\} – low prevalence | -1.153 | 5.109 | 14.355 | 87.188** |
| | (1.744) | (4.057) | (25.675) | (44.374) |
| $\mathbb{1}$\{Contagious diseases in $t-1$>0\} – high prevalence | -6.964** | 3.302 | -27.018 | 169.816*** |
| | (3.406) | (3.597) | (26.835) | (59.895) |
| $\mathbb{1}$\{Non-contagious diseases in $t-1$>0\} | 5.726** | 8.659** | -0.824 | 59.295 |
| | (2.799) | (3.892) | (28.816) | (51.647) |
| R-squared | 0.276 | 0.258 | 0.196 | 0.179 |
| Observations | 341 | 356 | 561 | 389 |
| District and Period F.E. | Yes | Yes | Yes | Yes |
| Mean investment | 5.37 | 19.30 | 100.44 | 154.15 |

See Table 1.1 for notes.

**Table A-1.5:** Other inputs' determinants - across two periods

| | Teachers per 1,000 capita | | Nb Conscripts per 1,000 capita | | Public Works per capita | |
|---|---|---|---|---|---|---|
| | **1908-31** | **1932-48** | **1908-31** | **1932-58** | **1908-31** | **1932-48** |
| **The structure of the colonial system:** | | | | | | |
| $\mathbb{1}$\{Medical center in $t-1$>0\} | -0.259 | -3.782* | 0.205 | -0.996 | 3.343* | -1.878 |
| | (0.422) | (2.190) | (0.236) | (0.781) | (1.860) | (2.275) |
| Colonial staff in $t-1$ | 0.058** | 0.068 | 0.034** | 0.253*** | 0.054 | 0.092** |
| | (0.024) | (0.087) | (0.017) | (0.053) | (0.135) | (0.045) |
| Strong presence of health adm. in $t-1$ | 0.648 | 3.761 | 0.075 | -0.638 | -4.400* | 1.421 |
| | (0.643) | (2.963) | (0.198) | (1.337) | (2.510) | (1.745) |
| Strong presence of other adm. in $t-1$ | 1.125** | 11.502*** | 1.046** | -35.333 | -5.029** | 14.268 |
| | (0.441) | (2.296) | (0.412) | (27.067) | (2.369) | (9.780) |
| **Context:** | | | | | | |
| $\mathbb{1}$\{Cities in $t-1$\} | 2.138* | 2.556*** | -0.071 | 0.544 | 8.404** | 0.560 |
| | (1.237) | (0.832) | (0.358) | (0.957) | (4.135) | (1.309) |
| **Diseases:** | | | | | | |
| $\mathbb{1}$\{Contagious diseases in $t-1$>0\} – low prevalence | 0.672 | -0.737 | -0.139 | -2.229 | 0.369 | 0.240 |
| | (0.540) | (3.229) | (0.346) | (1.461) | (1.683) | (1.832) |
| $\mathbb{1}$\{Contagious diseases in $t-1$>0\} – high prevalence | 1.924** | -1.598 | -0.017 | -1.534 | -0.013 | 1.789 |
| | (0.764) | (3.102) | (0.374) | (1.539) | (1.870) | (2.311) |
| $\mathbb{1}$\{Non-contagious diseases in $t-1$>0\} | -0.496 | 1.826 | 0.517 | 2.316* | -1.782 | 6.283** |
| | (0.546) | (2.786) | (0.434) | (1.203) | (1.423) | (2.587) |
| R-squared | 0.274 | 0.277 | 0.232 | 0.326 | 0.215 | 0.452 |
| Observations | 398 | 258 | 725 | 893 | 184 | 204 |
| District and Period F.E. | Yes | Yes | Yes | Yes | Yes | Yes |
| Mean investment | 4.16 | 7.34 | 1.75 | 4.56 | 11.45 | 9.81 |

See Table 1.1 for notes.

# CHAPTER 2

## THE DOUBLE AFRICAN PARADOX: HEIGHT AND SELECTIVE MORTALITY IN WEST AFRICA

**Abstract:** This paper studies the relationship between adult height and under-five mortality in the context of the "double African Paradox". Africans are relatively tall in spite of extremely unfavorable income and disease environments. Moreover, their height stature decreased since the 1960's despite improving health conditions and a fall in under-five mortality rates. This study points to selective mortality by bringing forward a positive correlation between mothers' height and mortality in the 1980's West Africa. It then estimates a new model of height differential between survivors and deceased. Results imply that selective mortality could be large enough to mask significant height increases in the 1980's West Africa. In high mortality contexts, anthropometric studies should discuss mortality levels and trends to account for selective mortality. More generally, results imply that the issue of selective mortality is crucial to assess the long-term impact of most health interventions.

## 2.1 Introduction

Trends or differences in anthropometric measures are often used to approximate trends or differences in health standards. As a consequence, descriptive analyzes of health standards trends, as well as evaluations of health policies, often rely on measured child or adult heights.

Since works by Fogel (1994) , Steckel (2008), Komlos and Baten (2004), height has been very much used in development economics and economic history. It is a relevant health indicator because height evolutions are related to three components: genetics, pathogens and nutrition; the latter two during childhood (it is generally argued that height is mainly determined before age five). Hence, in a genetically constant population, height trends are explained by nutrition and health environment, the latter including both net exposition to pathogens and the health system. Furthermore, the study of height stature provides an insight into living conditions in contexts where no other well-being measures are available (see for instance Austin, Baten, and Moradi (2007), Moradi (2009) and Cogneau and Rouanet (2011)).

Height stature is very much used as an health indicator. It is key to understand the determinants of this health outcome and to investigate whether it partly reflects trends or differences in another key indicator of welfare, mortality. Most studies using anthropometrics treat their samples as if they were a representative sample of the birth cohorts that they consider. This assumption is not true if selective mortality induces a significant bias on mean heights. "If height is related to the risk of dying, changes in mortality will alter the distribution of heights in the adult population" (Alter, 2004).

This work relies on the definition of selection provided by Bozzoli, Deaton, and Quintana-Domeque (2009): "the indirect positive effect that comes from mortality selectively removing the least healthy (or shorter) members of the population, so that the survivors are healthier (or taller)". The critical question here is whether selective mortality is such that it can bias anthropometric studies. This question has already been raised by Bodenhorn, Guinnane, and Mroz (2013). According to these authors, "the historical heights literature often relies on micro-samples drawn from sub-populations that are themselves selected". Using micro-data on British soldiers' heights in the late eighteenth and nineteenth centuries, they show strong evidence of selection. They conclude that "observed heights could be predominantly determined by the process determining selection into the sample".

It is critical to estimate the extent of selective mortality in the sub-Saharan context, because of two puzzling stylized facts. This paper calls these two facts the "double African paradox". First, Africans are relatively tall in spite of extremely unfavorable incomes and disease environments, in particular, in spite of very high under-five mortality rates. This is the "African

Paradox" put forward by Bozzoli *et al.* (2009),[1] called in this paper the "level paradox". Second, under-five mortality rates decreased very quickly in sub-Saharan Africa during the last decades, which reveals that childhood health conditions improved. Yet, for the same cohorts, adult height stature, another proxy for health conditions during childhood, decreased in the very same region.[2] These descriptive results are puzzling because height and mortality are correlated in opposite ways with the same factors. Height is positively correlated with nutrition and negatively correlated with pathogens' prevalence. These correlations reverse for under-five mortality. Hence, one might expect a negative correlation between those two indicators; which is what is observed both in level and in trend outside of sub-Saharan Africa.

To sum up, the relationship between height and under-five mortality in sub-Saharan Africa is puzzling in two dimensions. It is the "double African paradox":

- A level paradox: Africans are relatively tall in spite of extremely unfavorable health conditions and low levels of income.
- A trend paradox: height stature did not increase during the last decades, even though under-five mortality rates significantly decreased, suggesting an improvement of child health conditions.

The question asked in this paper is whether selective mortality can explain, entirely or partly, this double paradox. Can the height advantage of African populations be explained by a mortality disadvantage? Can it be ruled out that selective mortality is large enough to mask height improvements, in a context where mortality rates have been declining rapidly?

## 2.2 Existing literature and contribution

The literature does not say much on the link between height and mortality, and even less in sub-Saharan Africa. So far, there is very few evidence that selection could induce a very significant bias in anthropometric studies. However, as raised by Bleakley (2010a): "the micro literature suggests that scarring effects are larger than selection effects, but the filter of publication bias might mean that we never see studies in which selection dominates scarring".

### 2.2.1 Literature

Studying a Norwegian cohort, Waaler (1984) shows that the likelihood of death between 40 and 69 years old is twice as large for shortest individuals as for tallest individuals. Starting from this result, Fogel (1994) makes the hypothesis that death probability is negatively correlated with height stature. He then correlates the decline in mortality rates in Europe during the

[1] African women are generally taller than Indian, Bangladeshi, and Nepali women in spite of much worse living conditions (Deaton, 2007).

[2] Adult height stagnated in the context of analysis of this paper, which is West Africa in the 1980's.

19th century with the increase in height stature. Alter (2004) brings together several studies and shows that the negative association between height and the risk of dying has been remarkably consistent in various contexts; numerous studies have shown that, within a given population, the probability to die was relatively higher for shorter people. This selective mortality implies that "when mortality is high, short people will be less likely to survive to adulthood. Consequently, those who become adults will be relatively taller" (Alter, 2004).

Two recent papers use the Chinese Great Famine (1959-1961) as a natural experiment to study the impact of a negative nutrition shock, inducing an increase in the number of deaths, on adult height stature. Gorgens, Meng, and Vaithianathan (2007) show that the famine induced 20 to 30 millions additional deaths among children and that survivors are not significantly shorter than the cohorts born just before the famine. This "non-result" stems from a selection effect (positive impact on adult height) and an undernutrition effect (negative impact on adult height). Showing that taller individuals were more lucky to survive during the famine, they demonstrate a "survival of the fittest" mechanism. Meng and Qian (2009) estimate the long-term impact of famine on survivors' height stature, education and occupation. The estimated effect of the famine is based on the survivors only and is biased downward. To overcome this bias, the authors look at the impact of the famine on the top decile (q90) of the height distribution. They also find that coefficients are significantly higher in absolute terms for the top decile. These two studies put forward a selective mortality mechanism. One might fear nonetheless that the results are context-specific: the authors exploit one of the most dramatic famine events in history.

Three papers provide a more general analysis of the relationship between height and infant mortality. Bozzoli *et al.* (2009) study the childhood determinants of adult height. In order to explain the level African paradox, the authors develop a model of selection and stunting. According to this model, the early life burden of nutrition and disease is responsible for mortality in childhood, which leads to selection. This early life burden also leaves a residue of long-term health risks for survivors (stunting), which has a negative impact on adult height. This model predicts that selection can dominate stunting at high enough mortality rates. In this work, the correlation between height and infant mortality is negative in sub-Saharan, but it is weaker there than in other part of the world.

Moradi (2010) focuses on the "level paradox" in Africa. He describes the extent of the survivorship bias on mean adult heights and asks whether the size is large enough to account for the African paradox. The first part of the paper uses cross-sectional data on developing and richer countries. The author finds that the effect of selective mortality can explain around one third of the level paradox. The second part of the paper builds on more micro data. Using

micro-data on Ethiopia and Gambia,[3] the author concludes that the survivorship bias is too small to explain the paradox. Rather, Moradi explains the paradox by the "growth catch-up" of African populations at puberty, by which Africans catch-up and end up 5 to 6 cm taller than they would have been otherwise. As discussed in Section 2.6.4, these findings are not in contradiction with the results of this work, depending on the interpretation of the growth catch-up.

Akachi and Canning (2010) start from the fact that health improving models we observed centuries ago in Europe, and more recently in developing countries, do not work in African countries. Using DHS data,[4] the authors investigate trends in infant mortality rates and adult heights in 39 developing countries since 1960. In sub-Saharan Africa only, despite declining infant mortality rates, adult heights have not increased. Elsewhere, improvements in nutrition, joint with health public policies, lead to a decrease in infant mortality rates, accompanied by an increase in height stature. The authors argue that health policies implemented in Africa had a focus on infections and diseases, which implied a direct decline in mortality, but not in morbidity. According to them, this explains why the mortality decline was not correlated with an height increase in this region. As will be discussed in Section 2.7.2, the results found by these authors could as well be explained by a decrease of selective mortality over time, which completely changes the conclusion in terms of health policies' impact.

Alderman, Lokshin, and Radyakin (2011) focus on the Indian context and document the impact of selective mortality on children's anthropometrics. Their theoretical framework is close from what is done in this paper. They simulate child height-for-age z-scores in the counterfactual scenario where all children would have survived until the survey. Depending on the assumptions they make, they estimate that the correction on 1992's child height levels would go from 0.1 to 0.8 cm.[5] They conclude that "although mortality risk is higher among malnourished children, selective mortality has only a minor impact on the measured nutritional status of children". The authors do not discuss the fact that even though the selection bias on z-scores levels is not substantial, trends might be significantly biased. From 1992 to 2005, observed survivors trends amount to +1.08 cm child height increases. Depending on the authors assumptions, simulated height increases go from +1.2 to +1.6 cm. The magnitude of these results is discussed in Section 2.6.4.

[3] Ethiopian Rural Household Surveys, and longitudinal surveys on heights from two Gambian villages, collected by Billewicz and McGregor (1982).

[4] DHS are Demographic and Health Surveys. These are the same data used in this paper, and described in Section 2.3.1.

[5] This equivalence is obtained by looking at the height-for-age z-score equivalence table for children aged one to three years old.

Several epidemiological studies also describe the link between malnutrition and mortality in sub-Saharan Africa, using anthropometric indicators (Billewicz and McGregor, 1982; Fawzi, Herrera, Spiegelman, Amin, Nestel, and Mohamed, 1997; Salama, Assefa, Talley, Spiegel, van der Veen, and Gotwa, 2001; Sterky, Mellander, and Wall, 1987). These studies generally focus on one country, or one region, and follow a given cohort of individuals using duration models. Such works aim at providing efficient public policies to fight against malnutrition in sub-Saharan Africa; they do not provide a complete view of the extent of selective mortality in the region.

### 2.2.2 Contribution

This paper studies the relationship between adult height and under-five mortality in the specific context of West Africa, in order to test for the existence of selective mortality. It focuses on adult heights rather than child heights with the aim for a complete view on the impact of selective mortality. If under-five mortality selects children that will *potentially* be taller at adult age but are not *actually* taller when children, selection could indeed impact adult heights more than child heights.

Identifying precisely the impact of selective mortality is not an easy task. Ideally, to identify a causal impact, mortality changes would need to be instrumented by a variable that impacts height only through selective mortality. It could be a natural experiment which would provide some exogenous variation in selective mortality that did not directly impact height. To that respect, standard instruments such as rainfall records, vaccination campaigns or conflicts cannot be used because they do not satisfy the exclusion restriction.

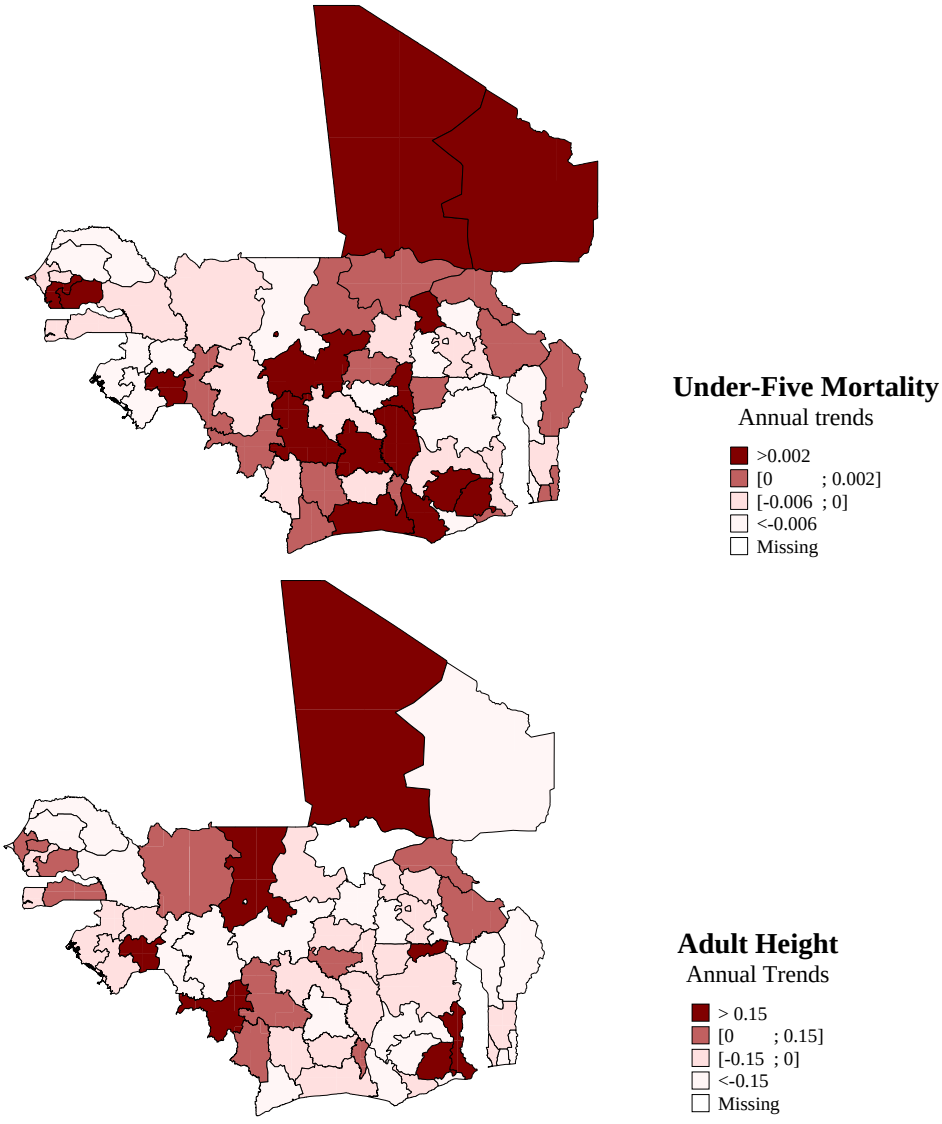
Consequently, the strategy developed in this paper is based on covariate changes in mortality, which concern every individuals in a given region. By using region rather than country fixed-effects, this paper better controls for the immediate health and nutrition environments, as well as for any unobservables that may impact height at the region level. This setting provides some evidence of a positive relationship between height and mortality in high mortality settings. To convince the reader that this result can be related to selective mortality, any other mechanisms that could explain such a positive correlation are ruled out.[6] Moreover, in order to document the extent of the selective mortality bias, this work builds an original model of the height differential between survivors and deceased.

The main contribution of this paper is to provide evidence that there is selective mortality on adult heights in the context of West Africa; and that it partially explains the "double African paradox". The maps in Figure 2.1 shows yearly evolutions for under-five mortality and adult height at the region level (using DHS data), in the 1980's West Africa. As this map illustrates, the correlation between adult height and under-five mortality is not unambiguously negative

[6] See a discussion on potential biases in Section 2.4.2.

in this setting. The magnitude of the selection bias estimated in this paper cannot give account of the level paradox. However, the results have major implications in terms of height trends' interpretation. It cannot be ruled out that selective mortality could mask adult heights trends of as much as $0.2$ cm per year in the 1980's West Africa. The fact that the selection bias could potentially reach such levels also has more general implications regarding the evaluation of health interventions.

**Figure 2.1:** Maps of West Africa: under-five mortality and adult height trends




Countries on the map: Benin, Burkina Faso, Cote d'Ivoire, Ghana, Guinea, Mali, Senegal. Annual weighted trends at the administrative region level, birth cohorts 1980-1991.

Section 2.3 presents the data used in this paper and some descriptive evidence. Section 2.4 puts forward a positive correlation between under-five mortality and adult height stature, in high-mortality regions. Section 2.5 builds a theoretical model for the height differential between survivors and deceased. The magnitude of the selection bias is estimated from this model, and is then confronted to the "double African paradox" in Section 2.6. The robustness of these results and their main implications are presented in Section 2.7. Section 2.8 concludes.

## 2.3 Data and descriptive results

### 2.3.1 Data

This work builds on DHS surveys (Demographic and Health Surveys) that were collected from 1992 to 2012 in Benin, Burkina Faso, Cote d'Ivoire, Ghana, Guinea, Mali and Senegal.[7] The analysis is restricted to West African countries covered by at least two geolocalized DHS surveys, containing mothers' heights.

Given that for each country, 3 to 4 surveys can be used, the pseudo-panel structure of the data is a great asset. The data allow to assign a given period and region to each observation. To do so, a geographical unit must be built, that would be constant from one survey to another. Only then, fixed-effects regressions can be implemented, in order to control for any unobservables defined at the regional level. To construct these fixed regions, DHS clusters are projected on the map of West Africa's administrative regions, and matched with them.[8]

DHS data are provided with survey weights that ensure the representativeness of the sample at the country level. However, sample size of surveys is not proportional to population size. In order to obtain a representative sample of the seven West African countries studied here, the whole sample is reweighed, taking into account the representativeness of each survey's sample.[9]

Each DHS survey contains stratified samples of mothers aged 15 to 49 who are asked about their reproductive history. In particular, information is given on the day of birth and death of each of their children, along with the child gender and mother's characteristics. Under-five mortality rates are computed at the region×cohort (periods of three birth years) level,[10]

[7] Surveys used in this paper are listed in Table A-2.1, in the Appendix.

[8] As a robustness check it can be verified that results are not driven by the definition of regions. Using an arbitrary grid (grid of squared zones, of side one and a half degree of longitude/latitude, with at least 5 clusters per zone and per survey year), results obtained are broadly unchanged, but they are less precisely estimated.

[9] Using World Bank population statistics, this work builds a sampling rate equal to the number of mothers in the survey implemented in country $j$ and year $i$, divided by the total population of country $j$ in year $i$. For each observation, the existing weight is divided by the corresponding sampling rate.

[10] This paper focuses on under-five mortality because the computation of this indicator is based on a larger number of individuals, which reduces the risk of measurement error. The computation of infant mortality is threatened by many measurement errors, in particular because mothers tend to round their children's death age to one. Results do not change if infant mortality (mortality before one) or child mortality (between one and five) are used as main

using age-specific death rates,[11] rather than crude death rate. Such a method controls for age distribution. Moreover, as it does not go back too far in birth histories, this method prevents from potential omissions' issues. Under-five mortality is computed on children born 60 to 180 months prior to the survey, i.e., those who have been fully exposed to the considered mortality risk.[12] The sample is also limited to children who have always lived in the surveyed village, to avoid potential migration biases. For each region×cohort, the mortality rate is equal to the share of those children who died before five. The average under-five mortality rate is 20%, but mortality rates can be as much as 40% in the beginning of the period.[13] Under-five mortality rates are computed at the administrative region level, pooling together three birth years into periods.[14]

All DHS surveys provide the height in cm of mothers aged 15 to 49.[15] In order to relate height of mothers to under-five mortality rates they actually faced as children, more recent surveys are used to compute mean mothers' heights and older surveys are used to compute mortality rates. Adult height is studied on a sample of mothers aged 20 to 49, assuming that height growth is achieved at 20.[16] A further assumption is that mothers' height levels and trends are representative of women's height levels and trends.[17]

Basic descriptive statistics on mothers' height and under-five mortality for three-year birth cohorts can be found in Table A-2.2 in the Appendix.

### 2.3.2 Descriptive statistics on the paradox

Table 2.1 provides descriptive statistics on the "double African paradox". In the sample, the average under-five mortality rate is 20%.[18] It means that around one out of five children dies before the age of five in the seven countries studied. At the same time, observed adult heights are relatively high. The region studied is characterized by both tall adult heights and high infant and child mortality rates. This is the level paradox.

explanatory variables.

[11] It is the ratio of the number of children dead before five to the number of births, for a *given birth cohort*. This rate approximates the probability to die before five in a given cohort.

[12] Results are unchanged when opening the window to children born five to 20 years before the survey.

[13] Results are robust to removing the few region×cohort where mortality reached such levels.

[14] The sample restricts on region×cohort observations using at least 30 eligible children to compute the mortality rate, i.e., at least 30 children born 60 to 180 months before the survey). This amounts to removing eight regions×cohorts in 1980's Guinea.

[15] More recent surveys also contain the height of non-mothers. To have a consistent sample of heights, this paper restricts to mothers' heights. Note that men are not measured.

[16] There is no clear consensus on the fact that height growth is completely achieved at 20. Consequently, all results control for a second-order polynomial of age to take into account the age composition of cohorts.

[17] This assumption has been verified for sub-saharan Africa in several anthropometric studies using DHS data. See for instance Cogneau and Rouanet (2011) on Cote d'Ivoire and Ghana.

[18] The infant mortality rate is 13%, and the neonatal mortality rate (before one month) is 6%.

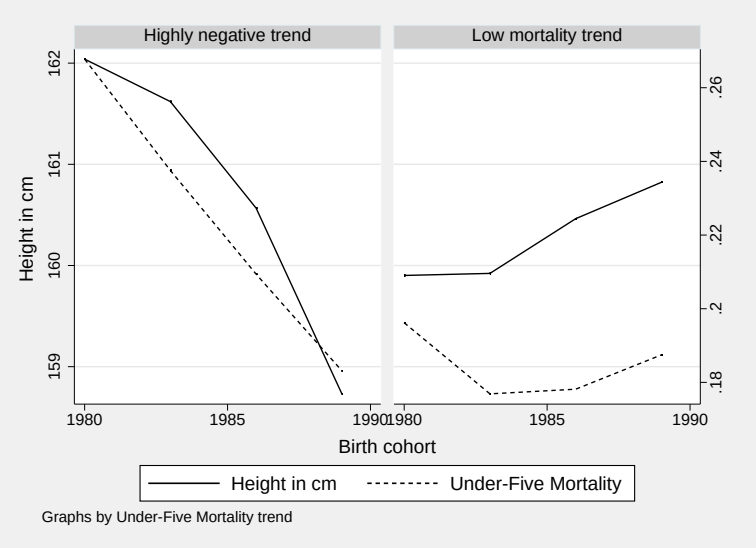
**Table 2.1:** Changes in height and mortality – the 1980's West Africa

| | Mothers 1980-1991 | | |
|---|---|---|---|
| **Indicator** | **Level** | **Trend** | **N** |
| Height stature (cm) | 161 | 0.007 | 16,773 |
| Under-five Mortality | 20% | -0.004*** | 84,446 |

Weighted regressions of individual mother height and regional mortality rate on birth cohorts. The height regression controls for age and age$^2$ because the age composition of mothers varies. N is the number of women (resp. children) on which height (resp. under-five mortality) statistics are computed. Sample of mothers: native mothers aged more than 20, born during a mortality cohort. Sample of children: native, 5 to 15 years old, born during an adult height cohort. Cohorts: 1980-91.

Table 2.1 also provides the magnitude of the trend paradox. For birth cohorts 1980-1991,[19] trends are estimated with region fixed-effects. Under-five mortality rates significantly decreased by 0.4 percentage point per year, while adult heights did not increase.[20] This is the trend paradox.

**Figure 2.2:** Height and under-five mortality trends - sample split in two types of regions




Same sample as in Table 2.1. Mean cohort effects estimates, with region fixed-effects. The sample is split by under-five mortality trend: (i) regions with highly negative mortality trend (under -0.6 percentage point per year, 40% of regions), (ii) regions with low or positive mortality trend.

In Figure 2.2, the sample of region×period is divided into two categories: highly negative under-five mortality trend (under -0.6 percentage point per year), and less negative or positive trend. This graph illustrates the fact that the adult height decrease is only significant in those regions with highly decreasing mortality trends. A decrease in height is only observed in regions facing a substantial mortality decrease (left graph), which are also regions starting with very high mortality rates.

[19] Birth cohorts 1975-1979 are excluded from the analysis for two main reasons. First, they were available for half of countries only. Second, for these older cohorts, mortality rates are computed on children born long before the survey, and are thus less reliable.

[20] The magnitude of the mortality decrease is comparable to one found by Akachi and Canning (2010), who find a decrease of 0.3 percentage point per year, for infant mortality.

**Figure 2.3:** Height distributions over two periods


Same sample as in Table 2.1. This figure graphs the univariate kernel density estimates of heights, using the Epanechnikov kernel function, over two periods.

Figure 2.3 shows height distributions for two birth cohorts of women: women born in 1980-1985 and born in 1986-1991. From 1980 to 1991, under-five mortality has been decreasing. Illustrating again the trend paradox, height distributions were shifted towards the left, during this same period. Furthermore, in the more recent period (cohorts 1986-1991), shorter women (less than 157 cm) appear in the distribution.

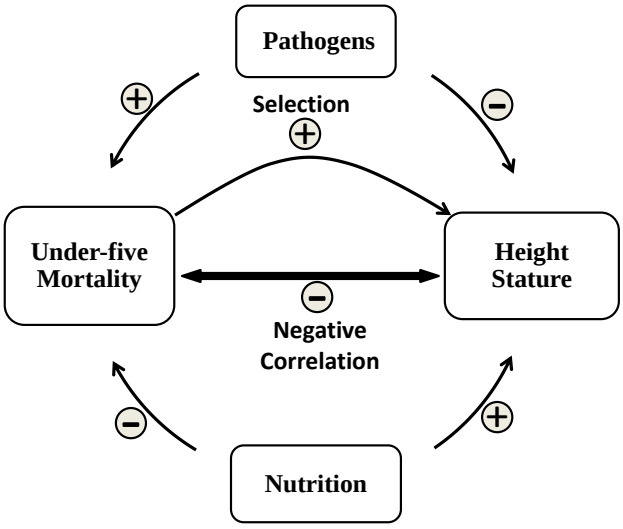
These descriptive results are coherent with a story of declining selective mortality, leading to the survival of shorter people in more recent cohorts. However, this story needs to be tested properly in order to assess whether selective mortality could induce a significant bias in anthropometric studies, and could explain both the level and the trend paradox. The following section estimates linearly and non-linearly the relationship between height and mortality, in order to test whether descriptive results are an artefact, or reveal a true positive correlation between both health indicators.

## 2.4 The height-mortality relationship

### 2.4.1 The mechanisms at stake

Shall we expect a positive or negative relationship between height and mortality? A monotonous one? The relationship between height and under-five mortality is a complex one, whose main simplified mechanisms are described in Figure 2.4.

**Figure 2.4:** The link between height and under-five mortality: main mechanisms




Crimmins and Finch (2006) use historical data from cohorts born before the 20th century in four northern European countries to study trends in longevity, age-specific mortality and adult height. They develop a heuristic model, which links height increases to decreases in infection, through reductions in inflammation: "if infections occur during development, substantial energy is reallocated at the expense of growth, as required by the body for immune defense reactions and for repair". They also recognize that other potential links between height and infections include improvements in diet and nutrition, and reductions in direct organ damage.

Deaton (2013) follows such works and insists that height stature is determined during childhood both by nutrition and exposure to pathogens, and hence directly linked to the health environment of individuals. Infant and child mortality are also determined by nutrition and infections. Nutrition has a negative impact on mortality because it provides "greater resistance to disease" (Alter, 2004), whereas higher exposure to pathogens is correlated with an increase in mortality rates. These patterns alone induce a negative relationship between height and mortality. Such a negative relationship is indeed observed outside of sub-Saharan Africa, both in level and in trend. However, this negative relationship is not unique: under-five mortality might have a direct positive impact on height stature through selection.

The impact of selective infant and child mortality on adult height amounts to a classic selection problem *à la* Heckman: the outcome, height, is only observed for individuals who were selected, i.e., who survived. However, given the structure of the DHS data, mortality selection on adult heights cannot be studied using standard selection models. The data provide observables for individuals who survived, but not for individuals who died. In this context,

the "heckit" estimation procedure would be robust only if it relied on a natural experiment, which would explain the probability to survive but not the observed height. Such a natural experiment does not exist for the present study.

This paper relies on another strategy. For a given amount of nutrition, bad health conditions have a negative impact on height, via stunting, and an indirect positive effect, via selection. Respectively, for a given amount of pathogens, nutrition has a direct positive impact on height and an indirect negative impact through selection. Hence, ideally, the proposed work should control for the amount of nutrition received and the exposition to pathogens, in order to get rid of any stunting mechanism and to isolate the selection mechanism. Such a complete dataset is not realistic. Given these data constraints, the first aim of the paper is to put forward a positive correlation between under-five mortality and height in the 1980's West Africa, and to rule out other mechanisms than selection; which is done in sections 2.4.2 and 2.4.4. Furthermore, Section 2.5 provides the reader with a theoretical model of the height differential between survivors and deceased.

### 2.4.2 What are the potential biases?

When regressing height on mortality, the identification relies on height differences that could come from some residual errors. If these errors are correlated with mortality, the estimated correlation between height and mortality can be biased. This section discusses the main potential biases, and how this paper deals with them.

First, one may think of a lot of shocks that could affect both under-five mortality and height stature. These shocks may be at the idiosyncratic or village level. It would be the case for instance of health policies, famines, climate shocks, epidemics, etc. Regional fixed-effects control for some of these shocks, but not for all. In a context where some households face a negative income shock that both increases the probability of death of their child and decreases their height stature, [21] the correlation between height and mortality might be underestimated. It is very likely that any of these shocks would affect mortality and height in opposite ways, and thus bias downward the results. In the context of this analysis, it is indeed difficult to imagine a shock that would increase both mortality and height over time.

Second, DHS data do not provide the place of birth. Hence women who emigrated from their place of birth cannot be followed. The estimation is carried out on native mothers only. It is generally acknowledged that taller women migrate more. Furthermore, people migrate more often to urban areas, where infant and child mortality rates are generally lower in the African context. In total, taller women would emigrate from high-mortality places to low-

[21] In the context of Cote d'Ivoire for instance, Cogneau and Jedwab (2012) find that cocoa growers decrease investments in children's human capital when they face a negative income shock due to the decrease of cocoa prices, which leads to a decrease in child height.

mortality places. Again, this bias would lead to an underestimation of the height-mortality correlation. Some evidence of the magnitude and sign of this bias is given in Section 2.7.1. As a first attempt to control for a migration bias, the complete set of control variables includes a proxy of the migration rate: the proportion of natives among mothers.

Third, as long as under-five mortality rates are on long-term trends, there could be a reverse causality bias with children of taller or shorter mothers having a lower mortality risk. The sign of the bias is discussed in Section 2.7.1. Furthermore, all estimations include the estimated height of the previous generation as a control variable.

Another potential bias could be that health policies mainly target under-five mortality. This targeting could be at the expanse of an improvement in nutrition. In regions where a huge effort was made on infant and child mortality decrease, long-term health and nutrition would not improve, due to a crowding out of other health policies. This policy specialization would drive the height-mortality correlation upward. Imperfect nutrition controls[22] are added in the estimation in order to account for such a mechanism.

Last, another mechanism that could lead to a positive link between under-five mortality and height stature is "sibling rivalry" (Moradi, 2010). Within the household, if the number of deaths decreases, the quantity of food per capita decreases as well. As a consequence, all things being equal, children might be more undernourished in low mortality contexts. However, in the specific context of the 1980's West Africa, both adults and children are taller in households where the risk of mortality within the household is smaller, which rules out this mechanism. One could also think of a similar pattern at the region level, which would then be closer to a Malthusian mechanism. At the region level, if the total quantity of nutrition stays the same, the fall in child mortality results in a decrease in per capita nutrition. This decrease in per capita nutrition could in turn lead to a decrease in observed adult heights, even in the absence of selective mortality. As an attempt to control for this, in addition to nutrition per capita, an adults to children ratio is built, and used as a control variable. This ratio is equal to the number of adult women (above 15 years old) per children ever born (less than 12 years ago).[23] It captures the age composition of the population.

Regarding the last two potential biases, each described mechanism would lead to a positive correlation between under-five mortality rates and per capita nutrition intakes, within districts (when under-five mortality decreases, nutrition decreases as well). Following the same specification as in the next section, and on the same sample, it can be shown that if anything,

[22] Nutrition controls are caloric, protein and fat intakes per capita, extracted from FAO country-level data. These are calculated from national consumption of each food item using nutritional tables of calorie, protein and fat content, and dividing by the population.

[23] As in Rossi and Rouanet (2015), I prefer to use the number of children ever born rather than the number of alive children, as it is more likely to be exogenous.

this correlation is negative.[24] This result is hard to reconcile with a policy specialization or a Malthusian story. Furthermore, the seven West African countries studied in this work were characterized by increasing per capita nutritional intakes over the 1980's. Again, this stylized fact is hard to reconcile with a decreasing nutrition story.

Compared to country fixed-effects, the region fixed-effects specification used in this paper mechanically lowers the magnitude of any bias defined at the region level. This may explain why this paper obtains, to my knowledge for the first time in the literature,[25] positive and significant coefficients of correlation between height stature and under-five mortality: results are "less" downward biased than existing works.

### 2.4.3 The empirical strategy

Individual data are aggregated into a panel of region×cohort.[26] As long as mortality can only be measured at the region level, it is more relevant to study the link between height and mortality at this same aggregated level. A panel of mothers' heights is obtained by keeping native mothers, defined as those who were born and lived in the same village until the survey.[27] These height data are matched to a panel of under-five mortality rates, which is obtained by keeping all native children. The sample is restricted to birth cohorts later than 1980. The following equation is estimated on this panel, with region fixed-effects:[28]

$$H^s_{zt} = \lambda M_{zt} + \alpha Y_{zt} + \xi V_{zt} + \chi C_{zt} + \omega N_{zt} + \rho R_{zt} + \beta A_{zt} + \gamma H^{moth}_{zt} + \nu_t + \epsilon_{zt} \qquad (2.1)$$

Where:

- $H^s_{zt}$ is the average adult height of surviving mothers born in region $z$ at time $t$.
- $M_{zt}$ is a measure of under-five mortality in region $z$, cohort $t$.
- $Y_{zt}$ is a vector of socioeconomic variables averaged over households living in $z$ at $t$.
- $V_{zt}$ measures the vaccination rate in region $z$ at $t$.
- $C_{zt}$ is the climate (rain and temperature) in region $z$ and year $t$.
- $N_{zt}$ is the proportion of natives among mothers.

[24] The regression of per capita kcal or protein intakes on under-five mortality, with region and year fixed-effects, leads to a negative correlation.

[25] Except for some epidemiological surveys that are run at a smaller scale than a country or a group of countries.

[26] See Section 2.3.1 for a definition of regions.

[27] All individuals for which the *native* variable is missing are also kept.

[28] There is an issue on whether standard errors should be clustered in this setting. As data are aggregated at the region level, residuals' auto-correlation within region is not an issue. Yet, error terms can be serially correlated, so that and standard errors are clustered at the region level.

- $R_{zt}$ is an adults to children ratio.
- $A_{zt}$ is a vector of mothers' average age and its square in region $z$, cohort $t$.
- $H_{zt}^{moth}$ is the inherited height level from the previous generation.
- $\nu_t$ is a set of birth cohort dummies (aggregated into three-year periods).

Adding birth cohort dummies amounts to controlling for the general trend. This is relevant as the mortality trend is significant over the period (see Table 2.1). The region fixed-effects setting is necessary because regions might be characterized by various levels of public health, access to health care, nutritional intakes or genetics. More generally, region fixed-effects allow one to get rid of any unobservable that might affect height stature or under-five mortality at a regional level. They take into account non-varying regional differences in height or mortality. "In Africa, there is enormous diversity of average heights across countries, presumably reflecting local nutritional, environmental, and disease conditions (or even genetic differences)" (Bozzoli *et al.*, 2009). The identification strategy thus relies on regions where under-five mortality has more or less decreased than the average. For these regions, $\lambda$ gives the conditional correlate of an under-five mortality change in terms of height.

**Control variables**

Ideally, the empirical strategy would control for parental (father's and mother's) height to capture the genetic determinants of height. Unfortunately, the data do not include any measure of father's height. Consequently, inherited height is proxied by the mean height of women who could have been the mothers of cohorts included in the sample.[29] On top of this inherited height, $H_{zt}^{moth}$, there are six sets of control variables, which will be referred to later as $X_{zt}$.

*1. The socioeconomic status ($Y_{zt}$)*

Height stature and under-five mortality are both correlated with the socioeconomic status through nutrition and pathogen. The data do not provide any information on household or individual nutritional intakes and exposition to pathogens. Hence, they are proxied by the the average socioeconomic status in the region.

The first control is the wealth index decile of the region×cohort.[30] This index is relevant to describe population or health dynamics (Hohmann and Garenne, 2009). A further control is

[29] These are women who had children born between 1980 and 1991, which are the birth cohorts used in the analysis. Their average height is computed by region×cohort.

[30] Following Hohmann and Garenne (2009), an absolute wealth index that is the sum of eight binary variables describing households' assets can be computed. The 8 items are: the household has electricity, owns a radio, a television, a refrigerator, a bike, a motorcycle, a car, the main floor material is neither sand nor clay nor dust.

the proportion of educated mothers in the region. Regarding housing environment, two control variables are added: the proportion of households who do not have access to toilet facilities and the proportion of urban households. Last socioeconomic controls are caloric, protein and fat intakes, extracted from FAO country-level data.[31]

*2. Vaccination rate ($V_{zt}$)*

DHS data provide information on the vaccination of every measured children. From there, an individual "vaccination degree" is computed, and then averaged at the region×cohort level.[32] This regional measure of vaccination can be used to proxy the health environment because it indicates the level of health care and the contagion risk in the region. Hence, the region×cohort vaccination quartile is used as a control.

*3. Climate variables ($C_{zt}$)*

This paper makes use of data from the Terrestrial Air Temperature and Precipitation: 1900-2008 Gridded Monthly Time Series, Version 2.01. These data extrapolate monthly precipitations and air temperature for a 0.5 degree grid. From these, averages over region×cohort are extracted: *(i)* mean rainfalls and air temperature during the growing season (April to July) in year $t$; *(ii)* standard deviations of rainfalls and air temperature over years $t$ to $t+4$ (first five years of life).

*4. Proportion of Natives ($N_{zt}$)*

The quartile of the proportion of natives among all mothers is added as a further control, to partly deal with an out-migration bias.

*5. Adults to Children ratio ($R_{zt}$)*

Another control is the quartile of an adults to children ratio, equal to the number of women (above 15 years old) per children aged less than twelve years old.

*6. Age controls ($A_{zt}$)*

A last control is a vector of mothers' average age and its square in region $z$, cohort $t$. These variables control for the fact that mothers' average age varies from one $(z,t)$ to another.

[31] For each individual, the average kcal, protein and fat amount received during the first five years of life is computed, according to her year and country of birth. Then, these individual intakes are averaged at the region×cohort level.

[32] For each child, eight dummy variables are created for: four polio injections, three DPT (diphtheria, pertussis and tetanus) injections and one tuberculosis injection.

### 2.4.4 A positive correlation?

Estimation results of Equation (2.1) are shown in Table 2.2. They cannot be interpreted as the causal impact of under-five mortality on height stature because mortality is not an exogenous variable for height stature. When looking at the height-mortality relationship, whatever the set of controls, the correlation is non significant. This non-result is driven by the opposite forces of the selection bias, and of omitted variables, among which health or nutrition shocks.

**Table 2.2:** Fixed-effect regression of mothers' height on under-five mortality

| | OLS | OLS |
|---|---|---|
| Under-five mortality | 0.690 | 2.362 |
| | (2.387) | (2.488) |
| **Controls:** | | |
| Region and survey Fixed Effects | Yes | Yes |
| Basic controls | Yes | Yes |
| Other Controls | | Yes |
| R2 | 0.359 | 0.402 |
| Observations | 230 | 230 |

Region fixed-effect regression. Sample: see Table 2.1. Complete regression results in Appendix, in Table A-2.3. Basic controls: age, age$^2$, socioeconomic status, vaccination, inherited height. Other controls: adults to children ratio, native ratio, climate.

The previously described "double African paradox" concerns Africa, the continent with the highest prevalence of child and infant mortality. Consequently, one is tempted to ask whether the selective mortality bias is a concern in high mortality settings only.

To test for this assumption, a spline regression of mothers' height on under-five mortality can be implemented, using 25%[33] as a unique knot and adding the usual controls. Results are shown in Table 2.3. The correlation is non significant in low mortality contexts, for mortality rates under 25%. In high-mortality contexts however, over this knot, the correlation is significant and positive.[34] The magnitude of the correlation implies that when under-five mortality rates fall from 35% to 25%, height decreases by around one centimeter.

**Table 2.3:** Spline regression of mothers' height on under-five mortality

| | |
|---|---|
| Under-five mortality$\leq$ 25% | -0.702 |
| | (2.970) |
| Under-five mortality$>$ 25% | 11.479** |
| | (4.448) |
| Region and Survey F.E. | Yes |
| Controls | Yes |
| R2 | 0.416 |
| Observations | 230 |

Spline regression, with region fixed-effects. Sample: see Table 2.1. Complete regression results in Appendix, in Table A-2.4. Controls: age, age$^2$, socioeconomic status, vaccination, inherited height, adults to children ratio, native ratio, climate.

[33] This knot was chosen by looking at the second order polynomial regression of height on mortality.
[34] Coefficients before and after the knot are different at 5%.

## 2.5 A model of height differential between survivors and deceased

A positive correlation between height and mortality can be exhibited in the specific context of West Africa in the 1980's, in high-mortality regions only. Finding a positive correlation between these two indicators points to a selection mechanism. This section develops a theoretical framework to describe more precisely how selective mortality interacts with adult height. A key concept of interest is introduced, the height differential between survivors and deceased.

### 2.5.1 The adult height equation

The first assumption of the model is that each individual $i$ is born with a potential adult height, which is the maximum level of height she can potentially reach with standard levels of nutrition and pathogens. This potential height is directly inherited from the potential height of the parents, ${H_i}^{moth}$.[35]

The *realized* adult height is explained by an inheritance factor, $\gamma$, plus individual covariates explaining height, $X_i$ (vector of socio-economic status, vaccination, climate, etc. defined in Section 2.4.3).

$$H_i = \gamma.{H_i}^{moth} + \alpha.X_i + \epsilon_i \tag{2.2}$$

The underlying assumption here is that the impact of all covariates adds linearly to the height potential. In this model, getting more nutrition or being exposed to less pathogens during childhood has a positive impact on the achieved height $H_i$. This assumption draws on existing works: "thus, even if food is not limiting, a reduction in the burden of infections will allow increased height and weight" (Crimmins and Finch, 2006).

Moving from an individual to a regional level, the mean height of native women born in region $z$ at period $t$ is predicted by:

$$H_{zt} = \gamma.{H_{zt}}^{moth} + \alpha.X_{zt} + \epsilon_{zt} \tag{2.3}$$

### 2.5.2 The height differential between survivors and deceased

$H_{zt}$ is not observed as such in the data. As some individuals die during childhood,[36] the adult height they reach is not observed. The data only provide the adult height of survivors $H^S_{zt}$, but not the adult height of deceased $H^D_{zt}$. As such, $H_{zt}$ is the counterfactual height that would prevail without mortality selection, which needs to be estimated. The model seeks to

[35] The potential height of parents being proxied by the height of the mother.

[36] The assumption here is that mortality between five and 49 is negligible and that the individuals only die between zero and five. The mortality bias could be reinforced if maternal mortality is higher where under-five mortality is higher as well (short birth intervals can be an underlying cause of both, for instance). This mortality selection on mothers would be transmitted to the next cohort through height inheritance. Using Günther and Harttgen's methodology, I find very low adult mortality rates and verify that their levels and trends are so low that they cannot explain the double paradox.

reconstruct what would be the distribution of height at each period $t$, if no children deaths had occurred. Height trends of the counterfactual height distribution would provide evidence on height trends, net from selection mechanisms. $H_{zt}$ is the weighted sum of survivors' height and dead people's height : $H_{zt} = H^S_{zt}(1 - M_{zt}) + H^D_{zt}M_{zt}$, where $M_{zt}$ is the under-five mortality rate in region $z$ at time $t$. From this:

$$H^S_{zt} = H_{zt} + \lambda_{zt}.M_{zt} \tag{2.4}$$

Where:

$$\lambda_{zt} = H^S_{zt} - H^D_{zt} \tag{2.5}$$

$\lambda_{zt}$ measures the difference between survivors and deceased heights, which is never observed. Taking $H_{zt}$ from Equation (2.3), Equation (2.4) is equivalent to:

$$H^S_{zt} = \gamma H^{moth}_{zt} + \alpha.X_{zt} + \lambda_{zt}.M_{zt} + \epsilon_{zt} \tag{2.6}$$

The height differential between survivors and deceased, $\lambda_{zt}$, can be modeled as follows:

$$\lambda_{zt} = \delta_0 + \delta_1.X_{zt} + \delta_2.M_{zt_0} \tag{2.7}$$

Where $M_{zt_0}$ is the initial mortality rate in region $z$. Here, the first assumption is that the height difference between survivors and deceased is equal to a constant $\delta_0$,[37] and then depends on the covariates $X_{zt}$. The second assumption is that it depends on the initial mortality level in the region.[38] There are at least two mechanisms that justify this assumption. First, in high mortality settings, the probability that mortality is partly caused by malnutrition is also higher, which would increase the height differential between survivors and deceased. Second, due to a Malthusian mechanism, when baseline mortality is higher, survivors are allocated a higher amount of resources, again increasing the height differential. By replacing $\lambda_{zt}$ in Equation (2.6):

$$H^S_{zt} = \gamma H^{moth}_{zt} + \alpha.X_{zt} + \delta_0.M_{zt} + \delta_1.X_{zt}.M_{zt} + \delta_2.M_{zt_0}.M_{zt} + \epsilon_{zt} \tag{2.8}$$

The estimation of Equation (2.8) gives $\widehat{\delta_0}$, $\widehat{\delta_1}$ and $\widehat{\delta_2}$. From Equation (2.7), these estimates can be used to compute $\lambda_{zt}$. The selection bias is assumed to come from two potential mechanisms: those who die earlier have a shorter potential height and those who die earlier are exposed to less nutrition and more pathogens during childhood.

[37] This constant could be interpreted as a constant differential height potential between survivors and deceased.

[38] The assumption that $\lambda_{zt}$ depends linearly on under-five mortality can be relaxed. For instance, it can be assumed that $\lambda_{zt} = \delta_0 + \delta_1.X_{zt} + \delta_2.M_{zt_0} + \delta_3.M^2_{zt_0}$. Results are unchanged under this assumption. Results are available on request.

$\lambda_{zt}.M_{zt}$ will give account of how much the level of height in region $z$ at time $t$ could be affected by selective mortality, given that from Equation (2.4), $H_{zt} = H^S_{zt} - \lambda_{zt}M_{zt}$.

### 2.5.3 Identification assumptions of the model

The validity of this model depends on the property of the error term, $\epsilon_{zt}$. In an ideal setting, $X_{zt}$ would be a complete set of controls for nutrition, pathogens, socioeconomic and health environment. In this setting, $\epsilon_{zt}$ would not be correlated to $X_{zt}$ and $X_{zt}.M_{zt}$ in Equation (2.8). Only then, $\lambda_{zt}.M_{zt}$ would provide the causal impact of selective mortality on observed adult heights.

However, the data do not offer this complete set of controls, and the properties of the error term have to be discussed. If the error term is correlated with $M_{zt}$, there is an induced bias on the estimation of $\lambda_{zt}$, because $\delta_0$ is not precisely estimated in Equation (2.8). This could be the case if, in Equation (2.3), the variables included in $X_{zt}$ do not encompass completely the link between height and mortality. Then, some of the impact of mortality would remain in the error term $\epsilon_{zt}$. As a consequence, $\lambda_{zt}$ would not be precisely estimated from Equation (2.7). Fortunately, the assumption can be made that the bias on $\delta_0$ is negative. This assumption holds as long as the average height of the whole population (survivors and deceased) correlates negatively with under-five mortality rates at the region level. Following the simplified mechanisms presented in Figure 2.4, this is likely to be the case. Under such plausible assumptions, $\lambda_{zt}$ would be underestimated.

Another threat could stem from multiplicative effects of $X_{zt}$ and $M_{zt}$. If the error term $\epsilon_{zt}$ correlates with $X_{zt}.M_{zt}$, then $\delta_1$ is not precisely estimated in Equation (2.8). It cannot be excluded that the bias on $\delta_1$, and thus on $\lambda_{zt}$, could be positive. Given these concerns, estimation results on $\lambda_{zt}$ should be interpreted with caution.

The magnitude of $\lambda_{zt}$ should be seen as a reachable impact of selection, rather than the true causal impact of selective mortality on height.

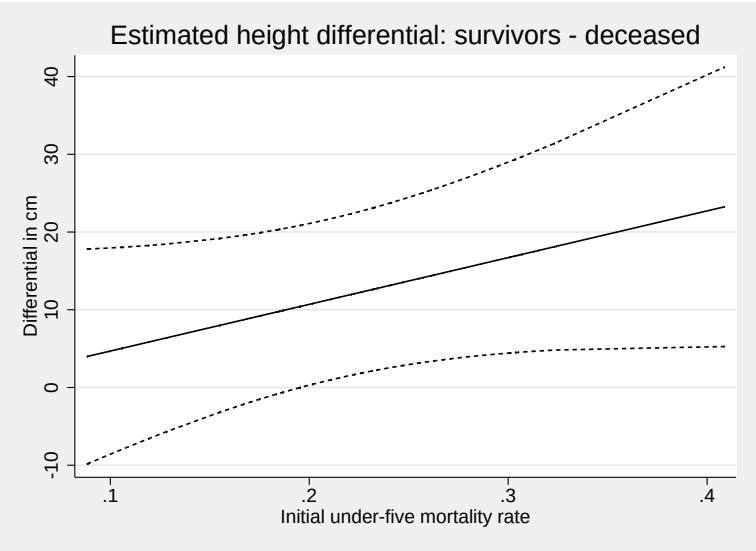
## 2.6 Estimation of the model and implication for the "double African paradox"

From the previous model, the potential extent of the selection bias in West Africa's 1980's can be assessed. A discussion is then made on the impact of such a bias on the "double African paradox".

### 2.6.1 Estimation of the height differential between survivors and deceased

Equation (2.8) is estimated on the same sample as in Section 2.4.4, again with region fixed-effects and cohort dummies. $X_{zt}$ is defined as the set of controls of Equation (2.1). $X_{zt} = (Y_{zt}, V_{zt}, C_{zt}, N_{zt}, R_{zt}, A_{zt})$. Estimation results are shown in the Appendix, in Table A-2.5. An estimate of the selection parameter, $\lambda_{zt} = \delta_0 + \delta_1.X_{zt} + \delta_2.M_{zt_0}$, is retrieved from these results.

**Figure 2.5:** Estimated height differential between survivors and deceased




Sample: see Table 2.1. This figure plots the estimates of $\lambda_{zt}$ against $M_{zt_0}$, obtained by inputing the results of Table A-2.5 in equation (2.8).

Figure 2.5 plots $\lambda_{zt}$ against the initial under-five mortality rate, $M_{zt_0}$. Two striking results are illustrated in this figure. First, $\lambda_{zt}$ increases with initial under-five mortality: $\delta_2$ is positive in Equation (2.7). Second, results imply that the height differential between survivors and deceased, $\bar{\lambda} = \frac{1}{N}\sum \lambda_{zt}$, could be as much as 12 cm. Furthermore, if the assumptions of the model are valid, this differential is significantly positive in contexts of high initial under-five mortality (above 20 to 25%). Again, what is estimated here is not the exact height differential between survivors and deceased. However, results imply that it cannot be ruled out that the selection bias could be as large as $\bar{\lambda}$. The magnitude of 12 centimeters cannot really be discussed in terms of the plausibility of its magnitude. By definition, the height of deceased, $H^D$, is never observed.[39]

[39] Yet, it has been shown that the variance of height is very high in a given population. In the sample of West African countries used in this paper, one can observe a significant economic gradient for height. All things being equal, women who are in the top quartiles of education, wealth, and protein consumption, and live in a urban area, with an access to modern toilet facilities, are more than three centimeters taller than others. In the 1920's to 1950's, several studies documented the height differential between the general population and very privileged, or selected, populations. Sutter, Izac, and Toan (1958), for instance, indicate that the average height of Polytechnicians was 175 cm in 1952, while the average height of the general male population in the 1950's France was 169 cm.

### 2.6.2 The level paradox

In the West African context, it cannot be ruled out that the height differential could be as much as 12 cm on average. Could the selection bias implied by this differential explain the height difference between Africa and Asia? Asian DHS surveys are used to answer this question.[40] For a given survey year, level of education and wealth, place of residence, year of birth and vaccination rate, Asian mothers are around 8 cm shorter than African mothers. The differential in under-five mortality rates between the two regions can explain $\lambda \times \Delta M = 0.8$ cm, given that the average differential in mortality between African and Asian countries is equal to 6.7 percentage point, and $\lambda = 12$.

These results imply that, under the assumptions made in this paper, at most 10% of the level paradox (an eight centimeter difference in height) could be explained by selective mortality. If African mothers had been exposed to the same mortality levels as Asian women, they could have been up to 0.8 cm shorter. In line with Moradi (2010) and Alderman *et al.* (2011), these findings imply that the magnitude of the selective mortality bias cannot explain massive differences in height levels. This paper agrees with Akachi and Canning (2010), who argue that "cross country differences in height are due to unobserved fixed factors". Another story has to be found to explain the structural height difference between African and Asian populations, and hence to solve the level paradox. Genetic differences might explain an important part of the difference, contrary to the statement of Deaton (2007) that "there are also good reasons for the generally prevailing view on the relative unimportance of genetic differences at the population level. [...] Americans of African descent (at least in large part) are as tall as Americans of Caucasian descent, and both are as tall as (most) contemporary Europeans".

### 2.6.3 The trend paradox

Can it be ruled out that selective mortality explains the trend paradox? $H_{zt}$ is an estimate of the adult height of the whole population, in the counterfactual scenario where all children would have survived. $H_{zt} = H^S_{zt} - \lambda_{zt}.M_{zt}$, can be retrieved from the estimate of $\lambda_{zt}$. If the assumptions of the model are true, $H_{zt}$ can be seen as a measure of height, corrected for mortality selection. It is thus called the "corrected" height. Table 2.4 shows linear trends of both the observed height and the "corrected" height. As already shown in Table 2.1, column (1) implies that the observed adult height did not increase over the 1980's. Column (2) shows that selection could be the sole driver of this non-increase. Indeed, the mean "corrected" height did increase, by 0.22 cm per year. In columns (3) and (4), the birth cohort is interacted with a dummy equal to one if the region experienced highly decreasing mortality trends, lower than

[40] DHS data for Bangladesh, Nepal and Cambodia are used. These are the three countries with at least two available geocoded surveys.

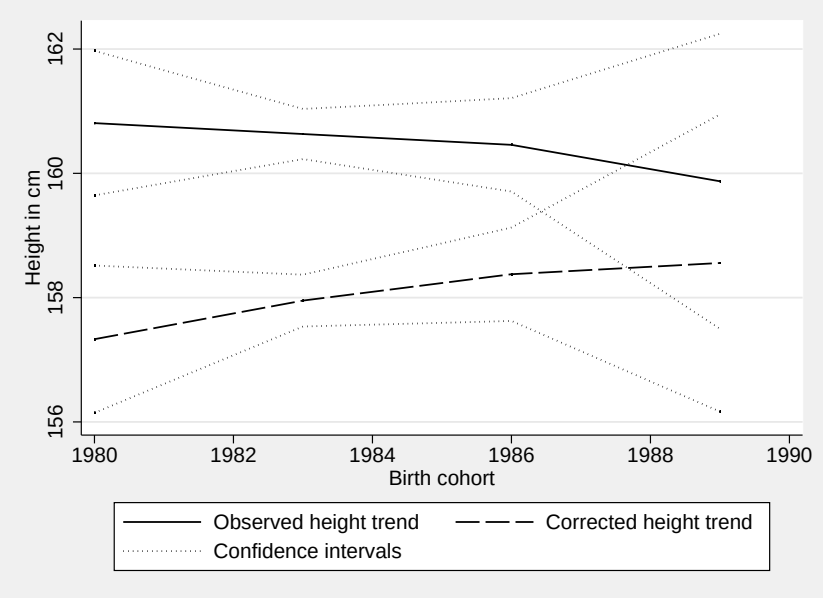
-0.6 percentage point per year. Column (4)'s results imply that the increase in "corrected" height was twice as large in regions with highly decreasing mortality as in other regions, and that the increase was only significant there.

**Table 2.4:** Observed and "corrected" height: linear trends

| | (1) Observed Height | (2) "Corrected" Height | (3) Observed Height | (4) "Corrected" Height |
|---|---|---|---|---|
| Birth cohort | -0.004 | 0.219* | 0.001 | 0.111 |
| | (0.121) | (0.117) | (0.156) | (0.156) |
| Birth cohort × | | | -0.005 | 0.120* |
| Mortality trend<-0.006 | | | (0.067) | (0.068) |
| pvalue of the sum | | | 0.973 | 0.064 |
| R-squared | 0.836 | 0.877 | 0.836 | 0.881 |
| Observations | 230 | 230 | 230 | 230 |

Sample: see Table 2.1. Observed height: adult height of survivors, observed in the data. "Corrected" height: estimated height of the whole population (survivors+deceased).

**Figure 2.6:** The trend paradox: what does correcting for selection imply?




Sample: see Table 2.1. This figure plots cohort dummies estimates for height obtained with region fixed-effects regressions. Cohort dummies' estimates and their confidence intervals are connected. The plain line plots the trend of the observed height of survivors, $H^S_{zt}$, while the dashed line plots the trend of the "corrected" height, $H_{zt}$, i.e. the estimated height of the whole population.

Another way to look at this is to plot the trends of observed height and "corrected" height, as in Figure 2.6. Correcting height for the selection bias changes the conclusion on adult height trends during the 1980's. The selective mortality bias could be large enough to predict that if under-five mortality had not fallen during the 1980's, height would have increased, rather than stagnated. As a consequence, the trend paradox could be entirely driven by mortality selection. These results also have more general implications, which are discussed in Section 2.7.2.

### 2.6.4 Adult heights, child heights: why do conclusions differ?

How to reconcile the present findings with the fact that results on mortality selection and child height are rather mixed in the existing literature? The main argument brought forward in this paper is that, during childhood, adults who are potentially taller are selected, but they are not substantially taller when children.

This is another interpretation of Moradi's "growth catch-up". Indeed, one specificity of sub-Saharan Africa is that there is a catch-up in observed heights between childhood and adulthood. This is also the case in the sample of regions used in this work. When a given birth cohort can be observed before and after adolescence, the height-for-age z-score is greater after puberty than before. According to Moradi (2010), this catch-up is linked to physical development around puberty. However, this paper argues that such a catch-up could rather be explained by selective mortality. In places where under-five mortality rates are high, selective mortality is such that only children with the highest height potential survive. Survivors are slightly taller as children, but the differential in adult height (never observed, as deceased never attain adult age) is even greater.[41] A further evidence of this mechanism is that the catch-up is greater in high-mortality settings.[42]

This also explains why this paper obtains a much larger selection bias than Alderman *et al.* (2011). As explained in Section 2.2.1, these authors focus on children's anthropometrics, for which the impact of selective mortality is expected to be lower than for adult heights. Furthermore, their work focuses on India, where the magnitude of the bias is also expected to be lower. Indeed, according to Alderman *et al.* (2011), "the impact of the imputations for height-for-age on the total sample is proportional to the mortality rates".[43] Moreover, this paper provides evidence that the survivorship bias increases non-linearly with mortality rates, in which case the bias would be even higher in the African context.[44]

To sum up, this paper argues that there is no "African paradox" regarding child heights because the child height differential between survivors and deceased is much smaller than the adult height differential.

[41] For instance, Billewicz and McGregor findings imply a 2 cm difference in child height between deceased and survivors, for mean heights around 80 cm. If the deceased had survived, this adult height differential would have been much higher, if only because mean height would have more than doubled from childhood.

[42] In a sub-sample of 100 region×cohorts, both girls and mothers are measured. When comparing mean young adult z-scores (15-23 years old), and mean girl z-scores (12-35 months old), adult z-scores are higher than girl z-scores. Furthermore, using the spline specification again, the growth catch-up between girls' and adults' z-scores increases with under-five mortality, in high-mortality contexts. Results are available on request.

[43] In 1992, the authors estimates an under-three mortality of 8%. According to World Development Indicators data, the under-five mortality in India was 12% for this same year, and was as high as 17.6% in developing sub-Saharan Africa.

[44] To approximate the African setting, the authors provide a simulation where they change the surviving status of living children with lowest probabilities of survival to reach the mortality rate of 15%. However, it is not clear what assumption they make on the mortality trends. Hence, unfortunately, no comments can be made on the simulated z-score trends they obtain.

## 2.7 Robustness of the results and main implications

### 2.7.1 Robustness

**Dealing with the migration bias**

This paper's results concern mothers who always lived in the village in which they were surveyed. This may imply a sample selection bias if women who left the village are specific and moved to specific areas in terms of mortality.

To get a sense of how the results would be affected if migrant women were included in the sample, one can make use of the Living Standard Measurement Surveys (LSMS) on Cote d'Ivoire (1985-1988) and Ghana (1987-1989). These household surveys share the unique feature of having measured the height stature of both adult men and women and of collecting very precious pieces of information on individuals' district of birth, allowing for the identification of migration. Under-five mortality rates are computed at the district of birth level, using the same methodology as with DHS data.[45] A drawback of these data, however, is that they cover a much shorter time horizon than DHS data. As a consequence, the exact mortality rate faced by adults cannot be computed. For this reason, the sample is restricted to men and women aged 20 to 30.[46] Their height is related to mortality rates prevailing 15 years after their birth.

In Table A-2.6, the stature of individuals who migrate is compared to the stature of individuals who did not, for a given district of birth. The first column shows that those who migrate are 0.65 cm taller than those who do not, which is in line with the existing literature. More interestingly, all this effect stems from districts with a high prevalence of mortality (last column).[47] In high-mortality contexts only, those who migrate are taller, by more than one centimeter. These results already give suggestive evidence that omitting migrants from the sample of analysis could induce a negative bias on the height-mortality correlation.

Table A-2.7 provides stronger evidence of this result. It shows the estimates of the conditional correlation between adult height and under-five mortality rate, using the closest specification as possible to Table 2.2. Adult height is regressed on under-five mortality, adding controls and region fixed-effects. Column (1) shows the estimate of this correlation on a restricted sample, focusing on non-migrants only, to approximate the DHS sample. Column (2) focuses on a larger sample, including migrants. Between column (1) and column (2), the conditional correlation moves from being significantly negative, to being non significantly different from zero. Omitting migrants from the analysis does induce a negative bias on the mortality coefficient $\lambda$ in Equation (2.1). This bias comes from the fact that excluding migrants from the

[45] The estimates obtained for mortality with LSMS data are smaller on average than those obtained with DHS data. LSMS surveys focus much less on demographic variables, so that there is probably much more underreporting of child deaths.

[46] Focusing only on women, the sample would be very small, with too few migrants to identify anything.

[47] High-mortality districts are defined as districts where the under-five mortality rate is above 12%.

sample amounts to removing tall people who were born in high-mortality areas, i.e. people who have a higher height-mortality correlation. Column (3) provides estimation results on the same sample as in column (2), but under-five mortality is interacted with a dummy equal to one if the individual has migrated from her place of birth. The coefficient in front of this interaction term gives the migrant specific mortality coefficient, $\Pi$. If migrants were included in the sample, the corrected mortality coefficient $\lambda^{corr}$ would be equal to:

$$\lambda^{corr} = (1 - mig_{zt})\lambda + mig_{zt}(\lambda + \Pi) = \lambda + mig_{zt}\Pi$$

Where $mig_{zt}$ if the migration rate of individuals born in region $z$ at time $t$.

The weighted average migration rate in Cote d'Ivoire and Ghana LSMS data is 58%.[48]. Thus, by adding migrants in the sample, the selection coefficient $\lambda$ is estimated to increase by $6.15 \times 60\% = 3.69$.[49]

If anything, the sample selection bias induced by omitting the migrants from the analysis leads to an underestimation of the mortality selection bias.

**Ruling out reverse causality**

The estimated conditional correlation between height and under-five mortality could be biased by a reverse causality going from height to mortality. This bias would be worrisome if taller mothers gave birth to weaker children. According to the work of Bhalotra and Rawlings (2011), looking at intergenerational health persistence, the opposite pattern is much more likely to be true. Looking at 38 developing countries over time, they find a negative and significant correlation between mother's height and under-five mortality. This result holds in the context of this paper. At the individual level, all things being equal, taller women are less likely to see their children die.[50] A reverse causality bias would thus lead to an underestimation of selective mortality.

### 2.7.2 Main implications

**Recent health trends in sub-Saharan Africa**

Akachi and Canning (2010) state that "in sub-Saharan Africa [...] while infant mortality has been falling, adult heights have been stagnating, or even declining, over the last 50 years. this undermines the view that since infant mortality rates have been falling, human capital has been rising". Their first assumption is that "broad based improvements in nutrition and public health measures" have an impact on both height stature and infant mortality. This explains

[48] In West African DHS data used in this paper, 60% of the sample do not live in its place of birth.

[49] This has to be compared to 11.5, the height-mortality coefficient in high mortality context, Table 2.3, or to $\bar{\lambda} = 12$, the mean estimate of $\lambda_{z,t}$, plotted in Figure 2.5.

[50] Results are available on request.

why, out of sub-Saharan Africa, the fall in infant mortality went hand in hand with an increase in height. However, they argue that in the specific context of sub-Saharan Africa, health interventions that reduced mortality had a "limited effect on disease prevalence, morbidity and physical development on children".

It is actually very hard to believe that health interventions had such a big impact on mortality trends, with a negative impact on the nutritional status. According to Stephensen (1999), many interventions exist to diminish the effect of infections on stature growth: "prevention of disease through sanitation, vector control, promotion of breast-feeding and vaccination is crucial". These are typically the sort of interventions that were led in Africa from the 1970's. Such interventions would improve the nutritional status of individuals, and hence their height. They would also decrease the prevalence of under-five mortality.

The results of this paper support the view that health interventions not only lengthen life spans, but may also have improved long-term health capital in Africa. In the sample of West African countries studied in this work, there is an increasing trend in nutritional intakes over the 1980's.[51] Moreover, the evidence of selection provided in this paper implies that a non-increase, or even a decrease, in adult height can be reconciled with a general improvement of health, including long-term health capital. From 1965 to 1980, under-five mortality fell from around 26% to 20%. Under the assumptions made in this paper, selective mortality could explain a -0.72 cm decline in adult heights ($12 \times 0.06$). Actually, the observed adult height trend for sub-Saharan Africa between 1965 and 1980 was -0.5 cm; mean height fell from 169.3 to 168.8 cm (Baten and Blum, 2014). It means that without the mechanical impact of selective mortality, observed adult height would have increased by +0.22 cm. To sum up, sub-Saharan African adults born in 1980 are formed by *(i)* individuals whose average health capital is slightly greater than that of cohorts born in 1965, but who would have survived even without increasing life spans; *(ii)* individuals whose average health capital is lower than that of cohorts born in 1965, who only survived thanks to the decrease in mortality prevalence. This surely could be interpreted as a general improvement of the population's health capital from 1965 to 1980. This conclusion is in contradiction with Akachi and Canning (2010).

It is in line with Alter's view that "if the selective effect of mortality on adult height is large, improvements in mortality may be confused with a worsening standard of living". Such confusion can be avoided by discussing the impact of the selective mortality bias.

[51] The quantity of calories and proteins per capita increases by 4% yearly, after adding the usual set of controls and region fixed-effects.

**The impact evaluation of health policies**

There is a much general concern for the impact evaluation of any shock which also has an impact on mortality. The most obvious shock having such characteristics is an health intervention which decreases mortality. $T$ is defined as a positive shock on health, occurring between period $0$ and period $1$. In such a setting, the impact of $T$ on an outcome $Y^S$ is estimated with a first-difference specification:[52]

$$Y_1^S - Y_0^S = \alpha + \beta T + \gamma Z + \epsilon \tag{2.9}$$

Where $Z$ is a vector of control variables. In Equation (2.9), $\beta$ is supposed to give the causal impact of $T$ on $Y$: $\mathbb{E}(Y_1^S - Y_0^S|T, Z) = \beta$.

At each period $t = \{0, 1\}$, $Y_t$ is defined as the outcome in the counterfactual scenario where mortality until adult age, or until the age at which $Y$ is observed, would be zero.

$$Y_t = Y_t^S(1 - M_t) + Y_t^D M_t = Y_t^S - (Y_t^S - Y_t^D)M_t \tag{2.10}$$

Where $Y_t^D$ is the outcome of deceased, and $M_t$ is the mortality rate. From Equation (2.10):

$$Y_1^S - Y_0^S = (Y_1 - Y_0) + [(M_1(Y_1^S - Y_1^D) - M_0(Y_0^S - Y_0^D)] \tag{2.11}$$

The impact of the health intervention $T$, net of mortality selection, is equal to $\mathbb{E}(Y_1 - Y_0|T, Z)$. When evaluating the impact of $T$ from $\mathbb{E}(Y_1^S - Y_0^S|T, Z)$, the selective mortality bias is equal to:

$$\mathbb{E}[(M_1(Y_1^S - Y_1^D) - M_0(Y_0^S - Y_0^D)|T, Z] \tag{2.12}$$

There are two scenarii in which this bias is null. First, when $Y^S = Y^D$, mortality is not selective. Second, when health interventions have an impact on morbidity rather than on mortality, they are not concerned by the issue of selective mortality.[53] It must also be acknowledged that selection is not an issue in studies looking at the direct impact of health interventions on mortality (Aslan and Goldin, 2015; Kesztenbaum and Rosenthal, 2014). Out of these scenarii, selective mortality can induce a significant bias on the impact evaluation of treatment $T$. What matters is the relative size of the bias, compared to $\mathbb{E}(Y_1 - Y_0|T, Z)$. From Equation (2.12), the magnitude of the bias increases with the change in mortality, and with the outcome differen-

[52] Similarly to the model presented in Section 2.5, $S$ stands for survivors. $Y_t^S$ is the observed outcome of surviving adults.

[53] See for instance Bleakley and Lange (2006), who use the Rockefeller-funded campaign against hookworm in the American South, around 1913. In this setting, hookworm was almost exclusively a childhood disease, with negligible effects on mortality.

tial between survivors and deceased. This is a less important concern in studies finding that $\mathbb{E}(Y_1^S - Y_0^S|T, Z) > 0$, in which at worst the impact of the shock is underestimated.[54] The selective mortality bias is more problematic in studies where $\mathbb{E}(Y_1^S - Y_0^S|T, Z) \leq 0$. For these studies, if the bias is large enough, not only the magnitude of the impact, but even its sign, could be biased. For instance, Acemoglu and Johnson (2007) study the impact of a change in life expectancy, on per-capita income, $Y^S$. They find that $\mathbb{E}(Y_{1980}^S - Y_{1940}^S|T, Z) < 0$. They also find that $\mathbb{E}(\overline{Y_{1980}^S} - \overline{Y_{1940}^S}|T, Z) \geq 0$, where $\overline{Y_t^S}$ is the total income in $t$. The authors do not discuss the fact that the difference between these two results could come from selective mortality. The decrease in income per capita found by Acemoglu and Johnson (2007) could indeed result from a selection effect, given that the fall in mortality was very large during this period.

The selective mortality bias can have important implications in terms of impact evaluation. It is not an issue in longitudinal studies, for which selective mortality cannot bias the conclusion that better health provides better outcome, for a given individual. However, for such micro-level studies, researchers face the same kind of complications when extrapolating their results to the general equilibrium, because of the health's effect on population size.

## 2.8 Conclusion

This paper studies the relationship between under-five mortality and height in the context of the "double African paradox" in West Africa; where adults are tall in spite of extremely unfavorable disease environments and adult heights decreased despite improving health conditions. Its originality is to focus on a region where under-five mortality rates are among the highest in the world, and to include region fixed-effects in the specification. This setting provides some evidence of a positive correlation between under-five mortality and adult height stature, in high-mortality regions.

As standard instrumentation or selection methods are not applicable in this context, a causal link between selection and height stature cannot be established using conditional correlations. Two strategies are developed. First, any other mechanisms that could explain such a positive correlation between adult height and under-five mortality, at the region level, are ruled out. Second, a new model of height differential between survivors and deceased is built and estimated.

Results obtained in this paper cannot give a good account of the level paradox. In line with the existing literature, this paper finds that the extent of selection is not massive, and only applies in high-mortality settings. As a result, another story than selective mortality has to be

[54] This is the case in Bleakley (2010b): "the most plausible composition effect would have reduced the income of the cohorts exposed to the eradication campaign as children". Similarly, the selection could not threaten the validity of Fogel's main conclusion that France and the U.K. underwent large increases in height stature.

developed to explain the persistent African adult height advantage.

This paper has important implications regarding the interpretation of height trends, and more generally regarding the impact evaluation of any health intervention that may affect mortality. The results of this paper are such that selective mortality could be large enough to mask significant height increases in the 1980's West Africa. In the absence of selective mortality in West Africa, adult heights could have increased by around $0.2$ cm per year rather than stagnated during the 1980's. This estimated trend is confirmed using several specifications. Hence, in high-mortality settings, the selective mortality bias can be such that survivors' height trends are misleading. By looking too quickly at under-five mortality trends, combined with survivors' height trends, one may conclude that health policies implemented in the 1980's in West Africa decreased the child mortality risk but did not improve long-term health. Such a conclusion would be biased, and partly driven by a selection bias on observed heights.

To conclude, any study finding that health interventions have a null or negative impact on long-term outcomes should discuss the selective mortality bias with great caution. This applies to studies looking at the impact of mortality-reducing health policies on long-term health outcomes or on incomes. In some settings, when the variation in mortality is large, it cannot be ruled out that the conclusion is not robust to a selection bias. Such settings are most probably past historical contexts, countries with very poor health, or large shocks on health (epidemic, famine, health interventions, etc.).

More generally, both theory and evidence suggest that we should stop thinking of health as a univariate object. Health's impact depends on how health changes. For instance a change in mortality, through its further selection effect, does not have the same impact as a change in morbidity. The health's impact also depends on when health changes (childhood, working age, or old age). Health is multidimensional, and must be treated as such.

# Appendix

**Table A-2.1:** DHS Surveys and birth cohorts used in the empirical part

| Surveyed Country | Survey Year | Birth Cohorts for : Mortality | | Height | |
|---|---|---|---|---|---|
| | | **Min** | **Max** | **Min** | **Max** |
| **Benin** | 1996 | 1981 | 1991 | | |
| | 2001 | 1986 | 1991 | 1980 | 1981 |
| | 2011 | | | 1980 | 1991 |
| **Burkina Faso** | 1992 | 1980 | 1988 | | |
| | 1998 | 1983 | 1990 | | |
| | 2003 | 1988 | 1990 | 1980 | 1983 |
| | 2010 | | | 1980 | 1990 |
| **Cote d'ivoire** | 1994 | 1980 | 1989 | | |
| | 1998 | 1983 | 1991 | | |
| | 2001 | | | 1980 | 1991 |
| **Ghana** | 1993 | 1980 | 1988 | | |
| | 1998 | 1983 | 1988 | | |
| | 2003 | 1988 | 1988 | 1980 | 1983 |
| | 2008 | | | 1980 | 1988 |
| **Guinea** | 1999 | 1984 | 1991 | | |
| | 2005 | 1990 | 1991 | 1980 | 1985 |
| | 2012 | | | 1980 | 1991 |
| **Mali** | 1995 | 1980 | 1986 | | |
| | 2001 | 1986 | 1986 | 1980 | 1981 |
| | 2006 | | | 1980 | 1986 |
| **Senegal** | 1992 | 1980 | 1988 | | |
| | 1997 | 1982 | 1991 | | |
| | 2001 | 1990 | 1991 | 1980 | 1985 |
| | 2005 | | | 1980 | 1991 |

This table lists all DHS surveys used in this paper , and the corresponding birth cohorts from which under-five mortality rates and mothers' height are computed.

**Table A-2.2:** Mean and standard deviations (SD) of under-five mortality and mothers' height, by country×period

| **Country** | **Birth Cohorts** | **Under-five Mortality** Mean | SD | **Mothers' Height** Mean | SD |
|---|---|---|---|---|---|
| Benin | 1980-82 | 0.234 | 0.046 | 159.6 | 6.6 |
| | 1983-85 | 0.197 | 0.028 | 160.1 | 6.5 |
| | 1986-88 | 0.198 | 0.016 | 159.1 | 6.6 |
| | 1989-91 | 0.187 | 0.019 | 158.7 | 6.7 |
| Burkina Faso | 1980-82 | 0.253 | 0.037 | 161.9 | 5.7 |
| | 1983-85 | 0.226 | 0.043 | 161.4 | 5.9 |
| | 1986-88 | 0.217 | 0.053 | 161.4 | 6.0 |
| | 1989-91 | 0.210 | 0.040 | 161.3 | 6.0 |
| Cote d'Ivoire | 1980-82 | 0.170 | 0.065 | 159.4 | 6.5 |
| | 1983-85 | 0.144 | 0.020 | 158.5 | 6.1 |
| | 1986-88 | 0.146 | 0.030 | 159.1 | 6.1 |
| | 1989-91 | 0.177 | 0.049 | 158.3 | 6.0 |
| Ghana | 1980-82 | 0.142 | 0.070 | 159.0 | 6.2 |
| | 1983-85 | 0.129 | 0.064 | 159.2 | 6.3 |
| | 1986-88 | 0.123 | 0.035 | 158.3 | 6.6 |
| Guinea | 1983 | 0.279 | 0.046 | 159.6 | 6.3 |
| | 1986-88 | 0.260 | 0.055 | 159.6 | 6.1 |
| | 1989-91 | 0.229 | 0.047 | 159.5 | 5.9 |
| Mali | 1980-82 | 0.275 | 0.080 | 161.1 | 6.0 |
| | 1983-85 | 0.271 | 0.054 | 161.3 | 6.3 |
| | 1986-88 | 0.248 | 0.049 | 160.3 | 6.2 |
| Senegal | 1980-82 | 0.198 | 0.034 | 163.3 | 6.3 |
| | 1983-85 | 0.180 | 0.032 | 163.8 | 6.7 |
| | 1986-88 | 0.160 | 0.030 | 163.4 | 6.5 |
| | 1989-91 | 0.156 | 0.030 | 162.5 | 6.1 |

**Table A-2.3:** Fixed-effects regression of mothers' height on under-five mortality
Full version of Table 2.2

[Cont'd next page]

| | | |
|---|---|---|
| Under-five Mortality | 0.690 | 2.362 |
| | (2.387) | (2.488) |
| **Basic Controls:** | | |
| Mother height | 0.051 | -0.082 |
| | (0.187) | (0.201) |
| Kcal | -0.003 | -0.002 |
| | (0.003) | (0.004) |
| Fat | 0.031 | -0.001 |
| | (0.028) | (0.036) |
| Protein | 0.109 | 0.121 |
| | (0.107) | (0.134) |
| Urban | 0.888 | 1.066 |
| | (1.369) | (1.434) |
| No Education | -1.537 | -0.882 |
| | (1.063) | (1.068) |
| Wealth index = 1 | 4.166 | 3.783 |
| | (2.731) | (2.731) |
| Wealth index = 2 | 1.463 | 1.125 |
| | (2.477) | (2.732) |
| Wealth index = 3 | 1.184 | 1.143 |
| | (2.597) | (2.836) |
| Wealth index = 4 | -1.164 | -2.357 |
| | (2.954) | (3.106) |
| Wealth index = 5 | 4.978* | 4.657 |
| | (2.713) | (3.005) |
| Wealth index = 6 | 7.291* | 6.222 |
| | (4.194) | (4.981) |
| Wealth index = 7 | 9.958* | 10.754** |
| | (5.122) | (5.244) |
| Wealth index = 8 | 24.716** | 24.633* |
| | (12.061) | (13.911) |
| Wealth index = missing | 10.500*** | 9.575** |
| | (3.503) | (3.667) |
| Access to toilet facilities | 0.183 | -0.127 |
| | (3.891) | (4.582) |
| Vaccination rate = 2 | -0.498 | -0.394 |
| | (0.441) | (0.477) |
| Vaccination rate = 3 | 0.353 | 0.370 |
| | (0.431) | (0.450) |
| Vaccination rate = 4 | -0.383 | -0.070 |
| | (0.445) | (0.428) |
| Vaccination rate = missing | 0.448 | 0.175 |
| | (0.579) | (0.678) |
| **Other Controls:** | | |
| Mean rainfalls | | -0.005 |
| | | (0.006) |
| Mean air temperature | | 0.076 |
| | | (0.359) |
| Standard deviations of rainfalls in $t$ to $t+4$ | | 0.020 |
| | | (0.013) |
| Standard deviations of air temperature in $t$ to $t+4$ | | 0.922 |
| | | (1.584) |
| Migration rate = 2 | | 0.222 |
| | | (0.394) |
| Migration rate = 3 | | -0.395 |
| | | (0.354) |
| Migration rate = 4 | | -0.783* |
| | | (0.409) |
| Migration rate = missing | | -0.500 |
| | | (0.569) |

| | | |
|---|---|---|
| Adults to children ratio = 2 | | 0.200 |
| | | (0.275) |
| Adults to children ratio = 3 | | 0.160 |
| | | (0.420) |
| Adults to children ratio = 4 | | 0.197 |
| | | (0.473) |
| Adults to children ratio = missing | | 0.422 |
| | | (0.662) |
| Age | -0.402 | 0.386 |
| | (1.124) | (1.692) |
| $Age^2$ | 0.008 | -0.008 |
| | (0.020) | (0.029) |
| Cohort=1983 | 0.045 | 0.082 |
| | (0.477) | (0.761) |
| Cohort=1986 | -0.226 | -0.355 |
| | (0.973) | (1.611) |
| Cohort=1989 | -0.699 | -0.514 |
| | (1.691) | (2.706) |
| Constant | 155.166*** | 160.311*** |
| | (33.484) | (42.078) |
| R2 | 0.359 | 0.402 |
| Observations | 230 | 230 |

*** p<0.01, ** p<0.05, * p<0.1, standard errors clustered at the region level. Under-five mortality rates are computed on children aged 5 to 15 y.o. Sample: see Table 2.1.

**Table A-2.4:** Spline regression of mothers' height on under-five mortality
Full version of Table 2.3

[Cont'd next page]

| | |
|---|---|
| Under-five mortality$\leq 25\%$ | -0.702 |
| | (2.970) |
| Under-five mortality$> 25\%$ | 11.479** |
| | (4.448) |
| **Basic Controls:** | |
| Mother height | -0.044 |
| | (0.204) |
| Kcal | -0.003 |
| | (0.003) |
| Fat | 0.007 |
| | (0.036) |
| Protein | 0.120 |
| | (0.133) |
| Urban | 1.142 |
| | (1.393) |
| No Education | -1.281 |
| | (1.120) |
| Wealth index = 1 | 3.489 |
| | (2.742) |
| Wealth index = 2 | 0.922 |
| | (2.727) |
| Wealth index = 3 | 0.959 |
| | (2.828) |
| Wealth index = 4 | -2.715 |
| | (3.095) |
| Wealth index = 5 | 4.372 |
| | (3.003) |
| Wealth index = 6 | 6.023 |
| | (4.881) |
| Wealth index = 7 | 9.948* |
| | (5.269) |
| Wealth index = 8 | 24.770* |
| | (13.524) |
| Wealth index = missing | 8.826** |
| | (3.622) |
| Access to toilet facilities | 0.326 |
| | (4.658) |
| Vaccination rate = 2 | -0.476 |
| | (0.481) |
| Vaccination rate = 3 | 0.342 |
| | (0.458) |
| Vaccination rate = 4 | -0.053 |
| | (0.457) |
| Vaccination rate = missing | 0.137 |
| | (0.677) |
| **Other Controls:** | |
| Mean rainfalls | -0.007 |
| | (0.007) |
| Mean air temperature | -0.025 |
| | (0.358) |
| Standard deviations of rainfalls in $t$ to $t+4$ | 0.024* |
| | (0.014) |
| Standard deviations of air temperature in $t$ to $t+4$ | 0.544 |
| | (1.506) |
| Migration rate = 2 | 0.247 |
| | (0.390) |
| Migration rate = 3 | -0.373 |
| | (0.364) |
| Migration rate = 4 | -0.739* |
| | (0.413) |
| Migration rate = missing | -0.572 |
| | (0.590) |

| | |
|---|---|
| Adults to children ratio = 2 | 0.216 |
| | (0.268) |
| Adults to children ratio = 3 | 0.097 |
| | (0.421) |
| Adults to children ratio = 4 | 0.096 |
| | (0.460) |
| Adults to children ratio = missing | 0.224 |
| | (0.678) |
| Age | 0.315 |
| | (1.697) |
| $Age^2$ | -0.005 |
| | (0.029) |
| Cohort=1983 | 0.164 |
| | (0.759) |
| Cohort=1986 | -0.137 |
| | (1.598) |
| Cohort=1989 | -0.120 |
| | (2.693) |
| Constant | 158.733*** |
| | (41.899) |
| R2 | 0.416 |
| Observations | 230 |

*** p<0.01, ** p<0.05, * p<0.1, standard errors clustered at the region level. Under-five mortality rates are computed on children aged 5 to 15 y.o. Sample: see Table 2.1.

**Table A-2.5:** Height differential model - Equation (2.8)
Fixed-effects regression of survivors' height at adult age ($H^S_{zt}$)

[Cont'd next pages]

| | |
|---|---|
| Under-five Mortality in $t$ $M_{zt}$) | 420.552* |
| | (233.464) |
| Under-five Mortality $\times$ Initial Mortality ($M_{zt}.M_{zt_0}$) | 60.111 |
| | (49.612) |
| Mother height | -0.014 |
| | (0.237) |
| $X_{zt}$ | |
| Kcal | -0.007 |
| | (0.005) |
| Fat | 0.331*** |
| | (0.119) |
| Protein | 0.140 |
| | (0.201) |
| Urban | 0.648 |
| | (3.383) |
| No Education | -0.236 |
| | (3.555) |
| Wealth index = 1 | 10.987 |
| | (9.508) |
| Wealth index = 2 | 10.387 |
| | (8.720) |
| Wealth index = 3 | 7.725 |
| | (10.775) |
| Wealth index = 4 | 1.157 |
| | (9.521) |
| Wealth index = 5 | 7.133 |
| | (9.827) |
| Wealth index = 6 | 17.052 |
| | (14.001) |
| Wealth index = 7 | -20.188 |
| | (15.028) |
| Wealth index = 8 | -80.058 |
| | (57.892) |
| Wealth index = missing | 14.876 |
| | (12.608) |
| Access to toilet facilities | 2.468 |
| | (5.494) |
| Vaccination rate = 2 | 0.223 |
| | (2.353) |
| Vaccination rate = 3 | 3.046 |
| | (2.014) |
| Vaccination rate = 4 | -220.710 |
| | (156.515) |
| Vaccination rate = missing | 4.464** |
| | (1.929) |
| Mean rainfalls | 0.034* |
| | (0.020) |
| Mean air temperature | 1.003 |
| | (0.710) |
| Standard deviations of rainfalls in $t$ to $t+4$ | -0.021 |
| | (0.021) |
| Standard deviations of air temperature in$t$ to $t+4$ | -3.533 |
| | (2.707) |
| Migration rate = 2 | 3.903** |
| | (1.493) |
| Migration rate = 3 | 3.333** |
| | (1.347) |
| Migration rate = 4 | 3.482 |
| | (2.097) |
| Migration rate = missing | 3.218 |
| | (2.110) |

| | |
|---|---|
| Adults to children ratio = 2 | 1.171 |
| | (1.099) |
| Adults to children ratio = 3 | -0.097 |
| | (1.524) |
| Adults to children ratio = 4 | -0.074 |
| | (1.575) |
| Adults to children ratio = missing | 0.917 |
| | (2.124) |
| Age | 2.473 |
| | (3.692) |
| Age$^2$ | -0.055 |
| | (0.071) |
| $X_{zt}.M_{zt}$ | |
| $M_{zt}$.Kcal | 0.032* |
| | (0.017) |
| $M_{zt}$.Fat | -1.445*** |
| | (0.521) |
| $M_{zt}$.Protein | -0.922 |
| | (0.557) |
| $M_{zt}$.Urban | -0.638 |
| | (15.510) |
| $M_{zt}$.No Education | -10.584 |
| | (17.061) |
| $M_{zt}$.Wealth index = 1 | -15.605 |
| | (39.322) |
| $M_{zt}$.Wealth index = 2 | -27.153 |
| | (34.627) |
| $M_{zt}$.Wealth index = 3 | -11.437 |
| | (44.247) |
| $M_{zt}$.Wealth index = 4 | 1.269 |
| | (41.286) |
| $M_{zt}$.Wealth index = 5 | -0.944 |
| | (42.953) |
| $M_{zt}$.Wealth index = 6 | -37.131 |
| | (66.616) |
| $M_{zt}$.Wealth index = 7 | 185.814** |
| | (89.101) |
| $M_{zt}$.Wealth index = 8 | 450.364** |
| | (224.415) |
| $M_{zt}$.Wealth index = missing | -25.468 |
| | (60.491) |
| $M_{zt}$.Access to toilet facilities | 0.945 |
| | (15.670) |
| $M_{zt}$.Vaccination rate = 2 | -4.558 |
| | (11.507) |
| $M_{zt}$.Vaccination rate = 3 | -14.481 |
| | (10.523) |
| $M_{zt}$.Vaccination rate = 4 | 1160.966 |
| | (823.166) |
| $M_{zt}$.Vaccination rate = missing | -18.802* |
| | (9.937) |
| $M_{zt}$.Mean rainfalls | -0.185** |
| | (0.080) |
| $M_{zt}$.Mean air temperature | -3.843 |
| | (2.876) |
| $M_{zt}$.Standard deviations of rainfalls in $t$ to $t+4$ | 0.281*** |
| | (0.103) |
| $M_{zt}$.Standard deviations of air temperature in $t$ to $t+4$ | 21.334** |
| | (10.018) |
| $M_{zt}$.Migration rate = 2 | -18.215** |
| | (7.257) |
| $M_{zt}$.Migration rate = 3 | -17.084** |
| | (6.960) |
| $M_{zt}$.Migration rate = 4 | -17.291* |
| | (9.044) |
| $M_{zt}$.Migration rate = missing | -12.591 |
| | (9.759) |
| $M_{zt}$.Adults to children ratio = 2 | -3.825 |
| | (5.781) |

| | |
|---|---|
| $M_{zt}$.Adults to children ratio = 3 | 2.548 |
| | (7.448) |
| $M_{zt}$.Adults to children ratio = 4 | 2.971 |
| | (7.717) |
| $M_{zt}$.Adults to children ratio = missing | 3.976 |
| | (9.677) |
| $M_{zt}$.Age | -22.920 |
| | (14.673) |
| $M_{zt}$.Age$^2$ | 0.465 |
| | (0.296) |
| Cohort=1983 | -0.095 |
| | (0.746) |
| Cohort=1986 | -0.453 |
| | (1.571) |
| Cohort=1989 | -1.274 |
| | (2.694) |
| Constant | 89.256 |
| | (76.030) |
| R2 | 0.640 |
| Observations | 230 |

*** p<0.01, ** p<0.05, * p<0.1, standard errors clustered at the region level. Under-five mortality rates are computed on children aged 5 to 15 y.o. Sample: see Table 2.1.

**Table A-2.6:** Selection into migration: regression of height on a migration dummy

| | **All regions** | **Low-mortality regions ≤12%** | **High-mortality regions >12%** |
|---|---|---|---|
| $\mathbb{1}${Migrant} | 0.647*** | -0.197 | 1.120*** |
| | (0.166) | (0.262) | (0.209) |
| Observations | 8,290 | 3,375 | 4,915 |
| R-squared | 0.459 | 0.479 | 0.450 |

*** p<0.01, ** p<0.05, * p<0.1, standard errors clustered at the region level. Data: LSMS surveys Cote d'Ivoire 1985-1988, Ghana 1988-1989. Sample: men and women aged 20 to 30. In the second and third columns, regions are split according to under-five mortality rates in birth departments. Controls: birth department dummies, survey year dummies, gender dummy. Migrant: has migrated from birth place, between her birth and the survey.

**Table A-2.7:** Impact of migration on the height-mortality correlation

| **Regression of height on birth department's under-five mortality rate** | | | |
|---|---|---|---|
| | **Non-Migrants only** | **All population** | |
| | (1) | (2) | (3) |
| Mortality rate | -3.697* | 0.427 | -2.879 |
| | (2.094) | (1.543) | (1.799) |
| Mortality rate × $\mathbb{1}${Migrant} | | | 6.150*** |
| | | | (1.969) |
| Observations | 5,078 | 8,290 | 8,290 |
| R-squared | 0.470 | 0.464 | 0.465 |

*** p<0.01, ** p<0.05, * p<0.1, standard errors clustered at the region level. Regression of adult heights on the under-five mortality rate prevailing 15 years after their birth (see text). Additional controls: education (literate, went to school), consumption, food expenditures. A dummy variable $\mathbb{1}${Migrant} is also included in the estimation. Control variables, sample and survey: see Table A-2.6.

# CHAPTER 3

## GENDER PREFERENCES IN AFRICA: A COMPARATIVE ANALYSIS OF FERTILITY CHOICES

*with Pauline Rossi*

**Abstract:** This paper proposes a new method to infer gender preferences from birth spacing. We apply it to Africa, where the least is known about gender preferences. We show that son preference is strong and increasing in North Africa. By contrast, most Sub-Saharan African countries display a preference for variety or no preference at all. Further analysis concludes that traditional family systems accurately predict the nature of gender preferences, while religion does not. Last, the magnitude of preferences is stronger for wealthier and more educated women.

## 3.1 Introduction

In the early 90s, Sen (1990) coined the term "missing women" to draw attention on the excess mortality of women in Asia: he estimated that approximately an extra hundred million women would be there if men and women received similar care in health, medicine, and nutrition. Since then, a large literature focusing mainly on South Asia and East Asia has described the discrimination against girls, mentioning for instance sex-selective abortions (Sen, 2001), differential child mortality (Rose, 1999), or differential health status (Pande, 2003). By contrast, Sub-Saharan Africa appears to do remarkably well. Sex ratios at birth are close to one, and survival rates as well as health outcomes are generally better for girls than for boys (Anderson and Ray, 2010; Wamani, Astrom, Peterson, Tumwine, and Tylleskar, 2007). All this may explain why gender preferences for children in Sub-Saharan Africa are rarely studied.

This paper focuses on fertility behavior as an alternative mechanism generating gender inequality, even when aggregate sex ratios are balanced. In their seminal paper, Ben-Porath and Welch (1976) infer the existence of gender preferences from the correlation between the probability to stop having children and the gender composition of existing ones. The idea has given rise to formal models of differential *stopping* behavior in favor of sons, predicting that an average girl has more siblings than an average boy[1] (Jensen, 2005). There might be important implications for gender inequality because girls would then face more competition for household resources.[2]

On the other hand, the analysis of differential *spacing* behavior is of special importance in the African context. Indeed, when couples have many children, gender preferences are more likely to lead to differences in birth intervals rather than in sibship size. Jensen (2005) and Basu and de Jong (2010) advocate looking at birth intervals to find evidence of gender preferences in a high-fertility context. Under son preference, differential spacing behavior implies that an average boy is breastfed longer than an average girl,[3] which may translate into inequality between boys and girls (Jayachandran and Kuziemko, 2011). Another reason to consider birth intervals is to account for health risks related to spacing, and not only to the number of births: according to the medical literature on developing countries, short birth intervals are associated

[1] As a simple example, consider a population in which parents have only one child if the first-born is a boy, and have two children if she is a girl. 50% of couples would have one son, 25% one girl and one son, and 25% two girls. Sex ratio is perfectly balanced at the aggregate level, but at the household level, girls have always one sibling whereas boys have, on average, one third of a sibling.

[2] Differential stopping behavior has been tested and validated by an extensive literature focusing again on Asia: e.g. Clark (2000), Jensen (2005), and Basu and de Jong (2010) in India, Abrevaya (2009) in the Chinese and Asian Indian populations living in the US, and Hatlebakk (2012) in Nepal.

[3] Again, as a simple example, consider a population in which parents always have two children; if the first-born is a girl, they try to have another child immediately, while they wait some time if the first-born is a boy. First-born girls, who represent half of the female population, are weaned prematurely while the entire male population is properly breastfed.

with adverse outcomes for mothers (Conde-Agudelo and Belizan, 2000) and children (Conde-Agudelo, Rosas-Bermudez, and Kafury-Goeta, 2006). The authors show that intervals lower than 24 months multiply the risk of infant death by 2.5, and intervals lower than 15 months multiply the risk of maternal death by two. If gender preferences turn out to induce short birth spacing, they could be a significant cause of maternal and infant mortality in Africa.

In this paper, we propose a new indicator of gender preferences based on differential birth spacing. We use a duration model of birth intervals to test if the gender composition of previous children influences the duration before the next birth. The main advantage of duration models is to deal properly with right-censored observations, i.e. families that are not yet complete by the time of the survey (Leung, 1988, 1991). We infer the existence of son (resp. daughter) preference when birth spacing is shorter for couples with fewer sons (resp. fewer daughters); and we deduce that preference for variety prevails when couples having a balanced mix of sons and daughters wait longer than couples having same-sex children. The conceptual framework underlying this strategy is a unitary model of the couple[4] choosing optimal spacing and stopping rules. People might have a taste for balance in the gender composition of children, or a girl/boy bias; then, costs and benefits may differ for sons and daughters. What is labeled as "gender preferences" is the outcome of a decision problem based on tastes and prices. Using duration models of birth intervals, son preference has been tested and validated in Asia,[5] but not in Africa. To our knowledge, there is no empirical study based on fertility behavior that documents systematically the variation in gender preferences in Africa. We contribute to fill in this gap using Demographic and Health Surveys in 37 African countries.

We find that, in North Africa (Morocco, Tunisia and Egypt), son preference is strong and has increased over time. By contrast, in most Sub-Saharan African countries, behavior is consistent with either preference for variety or no preference. South Africa, in particular, is characterized by a strong taste for balance. There is weak evidence of son preference in Mali, Senegal and in the Great Lakes region, but the impact on fertility patterns is not substantial. We further investigate the role of socioeconomic factors in shaping gender preferences. Wealthier and more educated women display the same type of preferences as the others, but the magnitude of their preferences is much larger. Then, in Sub-Saharan Africa, there is no correlation with religion: Muslims exhibit the same preferences as other religious groups. On the other hand, traditional kinship structure accurately predicts the nature of preferences: son preference prevails in pa-

[4]This is probably a strong assumption given the complexity of marital lives in Africa. In particular, we do not take into account that children might have different fathers, and that those fathers might also have children with other women. We will partly address this issue by comparing polygamous and monogamous women.

[5]E.g. in China (Tu, 1991), in Bangladesh (Rahman and DaVanzo., 1993), in the Chinese population of Malaysia (Pong, 1994), in Vietnam (Haughton and Haughton, 1995), in India (Arnold, Choe, and Roy., 1998), in South Korea (Larsen, Chung, and Gupta, 1998), and in Taiwan (Tsay and Chu, 2005).

trilineal ethnic groups only. Last, we discuss the different mechanisms through which gender preferences may translate into differential spacing. We conclude that our indicator mostly captures individual choices implemented through modern or traditional birth control methods.

The outline of the paper is as follows. Section 1 provides background on theoretical motives for gender preferences and a review of empirical evidence in Africa. Section 2 presents the data and some descriptive statistics. Section 3 discusses the empirical strategy and the identification assumptions. The main results are reported in Section 4, and some robustness tests are described in Section 5. Section 6 concludes.

## 3.2 Gender preferences in Africa

### 3.2.1 Theoretical motives for gender preferences

The most important motive put forward by the literature on gender preferences is the traditional structure of family systems. In patrilineal[6] and patrilocal[7] family systems, men are the fixed points in the social order, so that investment in daughters is considered as investment in another family's daughters-in-law. In Asia, such a system has produced economic incentives to have sons. For instance, the money spent for a son's marriage remains in the family while the dowry paid for a daughter's marriage is a net expense. In the same vein, female labor force participation is only valued once the daughter is adult, hence benefiting the family-in-law.[8] Last, sons act as old age insurance for their parents, because they are the ones who remain in the family's house. They also act as widowhood insurance for their mother, because widows' claims on the late husband's resources enjoy a higher social legitimacy if they have sons (Agarwal, 1994; Das Gupta, Zhenghua, Bohua, Zhenming, Chung, and Hwa-Ok, 2003). Mothers, in particular, really need a son because their status improves substantially when their sons get married: they can exert their power over daughters-in-law. Ultimately, women play a dramatic role in the perpetuation and reinforcement of patriarchy. Demographers working on Africa have come to similar conclusions (Lesthaeghe, 1989). Among the key factors shaping the reproductive regime in this region, they mention traditional inheritance patterns. In matrilineal societies, having daughters is necessary to perpetuate the lineage, whereas families need sons in patrilineal societies. But Africa is different from Asia along at least two dimensions. First, the system of brideprice prevails in almost all ethnic groups: the groom has to pay for the bride, contrary to what happens in a system of dowry. Second, the kinship structure is more flexible: adoptions and exceptions to allow daughters to inherit land in the absence of a son are

[6]Main assets are passed on through the male line whereas daughters are given movable goods.
[7]Upon marriage, wives move to their husbands' abode.
[8]In a context of child labor, son preference decreases when wages increase in the sectors of activity dedicated to girls (e.g. Koolwal (2007) in Nepal).

not unusual in African patrilineal societies. Eventually, the imperative to have a biological son is weaker in Africa than in Asia.

Another motive specific to Africa is the depth of Islamic penetration. In North Africa, the influence of the Islamic law is strong. These societies are characterized by property concentration, endogamous marriages and women seclusion, which implies that women's security and status critically depend on their ability to have sons. In Sub-Saharan Africa, traditions and customs have generally advocated common land ownership, exogamous marriages, women labor participation and women's societies, which renders women less dependent on their sons (Lesthaeghe, 1989).

The last part of the literature focuses on the impact of modernization factors on gender preferences; female education and labor participation, access to modern contraceptives, urbanization, economic growth and mass media are the most studied factors. The modernization hypothesis states that socioeconomic development would equalize the value of daughters and sons to their parents, leading to preferences for variety. However, modernization also brings about birth control - promoting smaller family size and facilitating sex-selective reproductive behavior - which could intensify, at least in the short run, traditional gender preferences. So far, the debate is still open, since empirical studies have found mixed results, depending on the context, the indicator, and the empirical specification they look at.[9]

### 3.2.2 Empirical evidence so far

In her review of the empirical evidence on gender preferences, Fuse (2008) concludes that, although North Africa has not been subject to much research compared to East or South Asia, there is evidence of strong gender bias against girls. She further writes that "of all sub-regions in the world, it appears that the least is known about Sub-Saharan Africa".

Cross-country analyzes generally find evidence of son preference in North Africa, but not in the rest of the continent (see Arnold (1992) on declared preferences and fertility behavior and Chakravarty (2012) on breastfeeding duration). Sub-Saharan Africa is characterized by a female advantage in infant mortality (Anderson and Ray, 2010), as well as in nutritional status and health outcomes (Wamani *et al.*, 2007). It does not display any systematic gender differences in breastfeeding and health seeking behavior (Garenne, 2003). Regarding declared preferences,[10] Fuse (2008) reports that most women in this region have no ideal gender composition, or would

[9] For instance, modernization factors are associated with smaller son preference in studies on India (Bhat and Zavier, 2003), Nepal (Barbar and Axinn, 2004), China (Arnold and Liu, 1986; Poston, 2002), or Egypt (Vignoli, 2006). But other studies on India (Basu, 1999; Das Gupta, 1987; Jayachandran, 2014; Rajan, Sudha, and Mohanachandran, 2000), South Korea (Edlund and Lee, 2013), Egypt (Yount, Langsten, and Hill, 2000) or Sub-Saharan Africa (Klasen, 1996) have questioned this result.

[10] Parents are supposed to have an ideal gender composition of children. Son (resp. daughter) preference is then defined as the ideal number of sons being strictly greater (resp. lower) than the ideal number of daughters.

prefer to have the same number of sons and daughters. As for household resources, Deaton (1987) found no evidence of differential allocation between boys and girls in Ivory Coast.

Still, some studies show that son preference may appear in Sub-Saharan Africa in case of income shocks. For instance, Flato and Kotsadam (2014) find that infant mortality increases more for girls than for boys during a drought; they further explain that such a difference is due to discrimination, since the effect is larger in communities more likely to discriminate against daughters (strong declared son preference, preference for a small family size and low female employment). In the same vein, Friedman and Schady (2012) find that girls are more exposed than boys to mortality risk in case of aggregate economic shock.
Last, a specific study on Nigeria shows that women with first-born daughters are significantly more likely to end up in a polygynous union, to be divorced, and to be the head of the household; they also have significantly more children (Milazzo, 2014).

Only a few papers estimate duration models of birth intervals in an African context. [11] Gangadharan and Maitra (2003) find evidence of son preference in South Africa, but only among the Indian community. Anthropologists working on the Gabbra, a patrilineal and patrilocal society in Kenya, find that women with no son have shorter birth intervals than women with at least one son (Mace and Sear, 1997). Last, Lambert and Rossi (2014) show that, in Senegal, women most at risk in case of widowhood substantially shorten birth spacing until they get a son. They relate son preference to women's needs for widowhood insurance.

Our paper contributes to the literature using duration models of birth intervals to test systematically for son preference in Africa. So far, evidence is quite limited in this region.

## 3.3 Data

### 3.3.1 Data

We use DHS surveys (Demographic and Health Surveys) that were collected from 1986 to 2012 in 37 African countries (surveys listed in Table A-3.1, in Appendix A). DHS data contain stratified samples of mothers aged 15 to 49 who are asked about their reproductive history. DHS data are provided with individual survey weights to ensure that the survey sample is representative of all mothers at the country level. Nonetheless, sample size of surveys is not proportional to population size. To obtain a representative sample of the 37 African countries studied, we reweighed the whole sample. [12]

[11] Some papers use duration models of birth intervals in Africa, but they are interested in the impact of socioeconomic factors (e.g. mother's characteristics such as birth cohort, age at first marriage and at first birth, residence, education in Ghilagaber and Gyimah (2004)), not in son preference.

[12] Using World Bank population statistics, we compute a sampling rate equal to the number of mothers in the survey implemented in country $j$ and year $i$, divided by the total population of country $j$ in year $i$. We also correct for the different number of surveys by country.

The main advantage of these data is that we observe all births, for children either alive or dead at the time of the survey, and we know the year and month of birth of all children, which enables us to measure birth intervals in months. Also, surveys are similar across countries, with a large number of observations (cf. Table A-3.1 in Appendix A), which makes possible a comparative analysis.

Nonetheless, the comparative analysis over space and time based on DHS data has two limits. On the one hand, surveys are not available in all African countries; notably Algeria, Libya, Mauritania, Eritrea, Somalia, Angola and Botswana are missing. Still, our sample represents 92% of the whole African population in 2009. On the other hand, the surveys took place during a relatively long period of time, so that by pooling the surveys together, we are considering different periods in different countries. In Sub-Saharan Africa, the period of interest is quite homogeneous: in all countries, the majority of mothers are born in the 60s-70s. This is true also in Egypt, but not in other North countries: in Tunisia, Morocco and Sudan, most women are born before 1960.[13] We have to keep these caveats in mind when interpreting cross-country comparisons and time evolutions.

We exploit a second source of data to get information on family systems. We use Murdock's data on African ethnic groups (Murdock, 1959) coded by Gray (1998)[14] to define which women belong to a matrilineal ethnic group. We opted for a conservative definition of matrilinearity, including only those ethnic groups listed by Gray that we found in the DHS data. Patrilinearity is identified by default, and probably includes some matrilineal groups.[15] Such a measurement error in our classification of ethnic groups is likely to flatten the differences between matrilineal and patrilineal groups. So when comparing the two categories, we estimate a lower bound of the difference.

### 3.3.2 Descriptive statistics

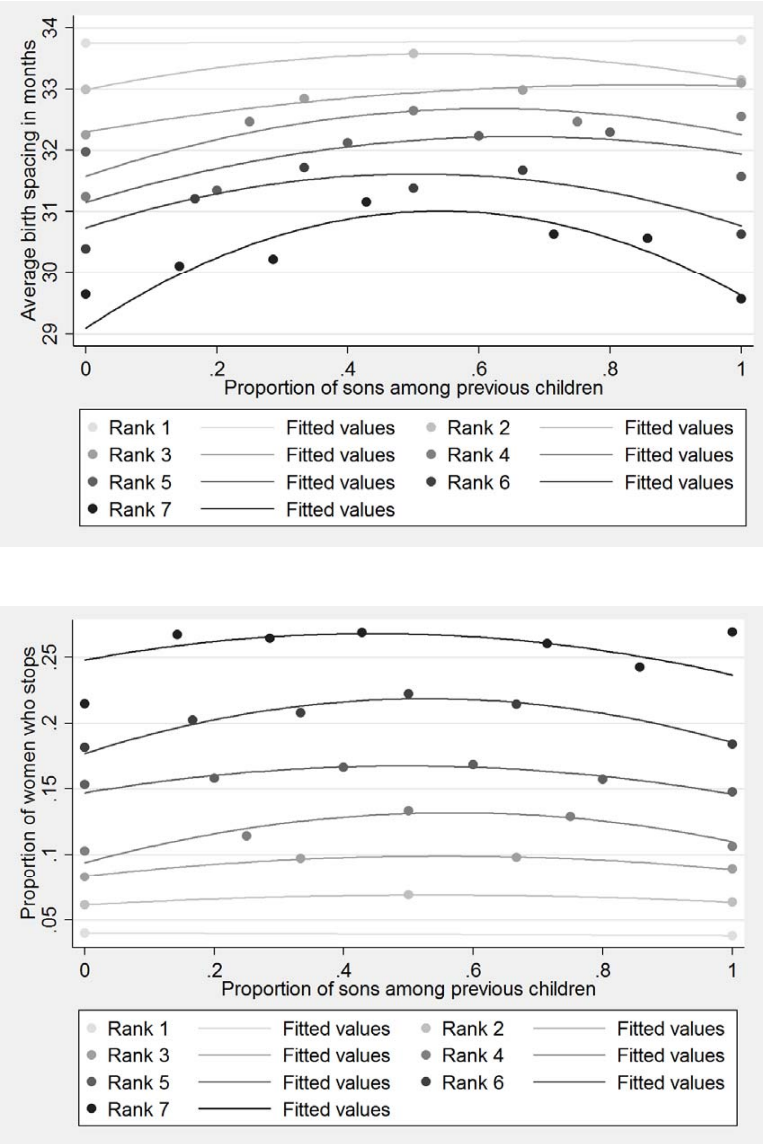
Before estimating the duration model, we provide some descriptive statistics of the non-censored durations. The upper graph in Figure 3.1 represents the average birth spacing by the gender composition of previous children. More precisely, we plot the average duration between births $n$ and $(n+1)$ as a function of the proportion of boys among the previous $n$

[13] On the other hand, the fact that we do not observe recent cohorts in Morocco and Tunisia makes our sample more homogeneous: all countries are at a very early stage of the fertility transition.

[14] Gray (1998) provides a database that lists the characteristics of African ethnic groups reported by Murdock. Of particular interest for us is the variable $Descent : MajorType$ that indicates whether an ethnic group is patrilineal or matrilineal. Table A-3.2, in Appendix A, gives details of our classification and explains how we matched Gray's and DHS data.

[15] We know from Gray (1998) that matrilineal ethnic groups exist in some countries, but the ethnicicty variable was missing in DHS data (e.g. Nigeria, Sudan, Tanzania, Zimbabwe) or DHS data types were too broad (e.g. DRC) so that we could not identify them.

**Figure 3.1:** Descriptive statistics on non-censored observations




Average duration between births $n$ and $(n+1)$ (upper graph) and proportion of women who stopped having children after the n$^{th}$ birth (lower graph) as a function of the proportion of boys among the previous $n$ children. Fitted values correspond to values predicted by a quadratic regression. Only ranks 1 to 7 are considered because at higher ranks, there are too few observations for each possible value of the proportion of boys. Lower graph: sample restricted to women over 40 years old to mitigate the issue of right-censoring.

children, for ranks one to seven. At each rank, birth spacing clearly displays an inverted U-shape:[16] it is lower for couples with no son or no daughter, and higher when the sex ratio is balanced. The maximum is reached by couples having slightly more boys than girls.

We find the same pattern when we look at the stopping behavior (cf. lower graph in Figure 3.1). We plot the proportion of women over 40 years old who stopped having children after the n$^{th}$ birth as a function of the proportion of sons among the previous $n$ children. Here again, women are more likely to stop having children when they already have a balanced mix of boys and girls.

In Appendix A, Table A-3.3 reports some statistics on fertility stopping and spacing behaviors, by country. In our sample, women have on average 6.2 children, the average birth interval is 35 months, and one third of birth intervals are shorter than 24 months. But there is a lot of variation across the continent. Southern African countries stand out because of long intervals and relatively low numbers of children: less than one fourth of short birth intervals and approximately four children per woman. The opposite is true for the Sahel region: between seven and eight children per woman, and more than one third of short birth intervals. In North Africa, the proportion of short intervals is over 40%, although the number of children is not that high, between five and six.

## 3.4 Empirical Strategy

### 3.4.1 A duration model of birth intervals

As explained in the introduction, we are mainly interested in differential spacing rules. We use a duration model of birth intervals to infer the existence of gender preferences. Our variable of interest $T$ is the duration between births $n$ and $(n+1)$, measured in months, where $n \geq 1$. Our coefficients of interest measure the impact of the gender composition of previous children on the subsequent birth interval. We estimate a Cox proportional hazard model (Cox, 1972).

The main reason to prefer duration models to linear models is the issue of censoring: the former allow us to identify the distribution of a duration variable from potentially right-censored observations if the duration and the right-censoring variables are independent. This condition is very likely to be satisfied as the date of the survey is completely unrelated to the latest births.

An alternative strategy would be to estimate, on the one hand, the probability to have another child, and on the other hand, the duration before the next birth. In our strategy, we implicitly assume that the impact of the gender composition on both decisions is the same. The first reason for this choice is parsimony: we want to build a unique indicator of gender pref-

[16] When we regress the non-censored durations on the proportion of boys and the proportion squared, coefficients are significant and of expected sign, whatever rank we consider. We further plot the lines corresponding to the quadratic regressions on the graph, and they fit quite well.

erences, in order to compare it across countries, periods, socioeconomic categories etc. Also, we would have to make some parametric assumptions to separate the stopping and spacing dimensions, while here, we are able to use a semi-parametric method of estimation. More importantly, in our context, it is not clear that fertility choices are a two-step decision process, in which people choose, first, if they want another child, and second, the timing of the birth. Cohen (1998) shows that couples in Sub-Saharan Africa use contraceptive to delay births rather than to limit them. If couples have more control over spacing out births than over stopping them, it may well be the case that they only decide to bring forward or to delay the next birth. The eventual number of births would then be mechanically determined by the successive decisions over timing together with the end of the couple's reproductive period.[17]

### 3.4.2 Relating durations to the proportion of sons

We want to design a model that exploits the information on all birth ranks, and not only intervals after a given rank. To do so, we create a variable $Frac_n$ equal to the proportion of boys among the previous $n$ born children.[18] We model the hazard function at each country level – the instantaneous probability to have another child at date $t$ – as follows:

$$\lambda(t) = \lambda_0(t) \times \exp(\alpha_1.Frac_n + \alpha_2.Frac_n^2 + \theta.X_n)$$

Where $\lambda_0(t)$ is the baseline hazard function, common to all individuals, and $X_n$ is a vector of mother's characteristics (birth cohort, age at birth $n$, age at birth $n$ squared, religion, family system, union type, education, wealth, area of residence, employment status);[19] it also includes a dummy for each rank $n$, to control for potential differences between birth orders. In our specification, the unit of observation is not the mother, but the birth. We reweighed the observations to ensure that each woman counts once, irrespective of her number of children.[20] We also use robust standard errors clustered at the woman level to account for the correlation between the error terms related to the different intervals of the same woman.

Under the proportional hazard assumption, $e^{\alpha_1+\alpha_2}$ measures the hazard ratio at any point in time between women having only sons vs. only daughters. If $\alpha_1 + \alpha_2 < 0$, having only sons vs. only daughters decreases the hazard rate and hence increases the expected birth interval. In

[17] As a robustness test, we also examine differential stopping rules, and as expected, we find much more scarce evidence of gender preferences (see Appendix B).

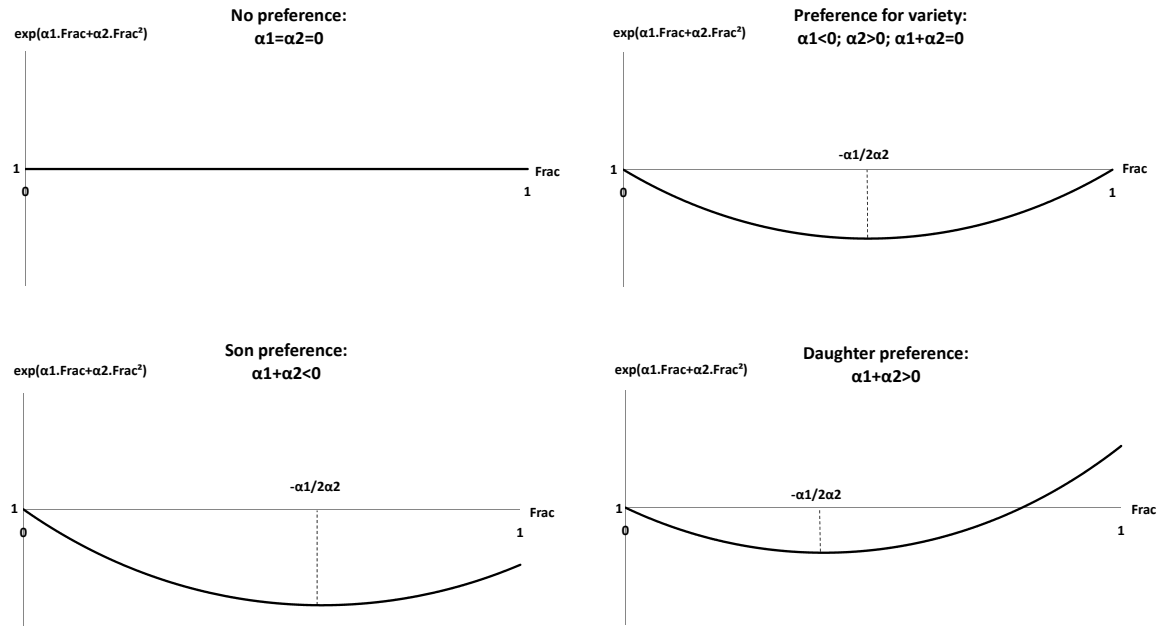
[18] In Section 3.6.3, we investigate whether revealed gender preferences differ across ranks. And in Appendix C, we show that our indicator is not only driven by couples wanting *at least one* son and/or one daughter, and that couples having one child of each gender keep displaying gender preferences.

[19] We introduce some controls to estimate more precisely the baseline hazard for different categories of mothers, thus reducing our standard errors. The magnitude of our estimates is unchanged if we remove the controls.

[20] We divide a woman's individual weight by her number of births. If we do not reweigh the observations, a woman with $n$ births counts $n$ times. So women having more children, meaning women with a taste for large families and older women, are over-represented.

this case, we infer the existence of son preference. Conversely, if $\alpha_1 + \alpha_2 > 0$, we infer daughter preference.

**Figure 3.2:** Multiplier on the baseline hazard as a function of the proportion of sons




$exp(\alpha_1.Frac + \alpha_2.Frac^2)$: multiplier on the baseline hazard.
$Frac$: proportion of sons among earlier births.
Each graph is calibrated for different values of $\alpha_1$ and $\alpha_2$ which are meant to represent an archetype of gender preferences. The lowest hazard rate, and therefore the longest duration, is predicted to be observed when $Frac = \frac{-\alpha_1}{2\alpha_2}$. We infer the nature of gender preferences by comparing this point to one half, which boils down the comparing $\alpha_1 + \alpha_2$ to zero.

We introduce the proportion squared to test for a taste for balance in the gender composition, as illustrated in Figure 3.2. We plot the multiplier on the baseline hazard as a function of $Frac$ for different values of $\alpha_1$ and $\alpha_2$. On the top left, we plot the trivial case in which $\alpha_1 = \alpha_2 = 0$, meaning that the gender composition of current children has no impact on subsequent durations. Then, if $\alpha_1 < 0$ and $\alpha_2 > 0$, it implies that the hazard rate is lower for women having children of each gender. The lowest hazard rate is reached by women having a proportion of boys among their children that is exactly equal to $-\frac{\alpha_1}{2\alpha_2}$. Therefore, the longest duration is predicted to be observed (i) among couples having exactly the same number of boys and girls if $\alpha_1 + \alpha_2 = 0$ (graph on the top right); (ii) among couples having sons and daughters, but more sons than daughters, if $\alpha_1 + \alpha_2 < 0$ (graph on the bottom left); and (iii) among couples having sons and daughters, but more daughters than sons, if $\alpha_1 + \alpha_2 > 0$ (graph on the bottom right). Our statistic of interest is therefore $(\alpha_1 + \alpha_2)$.

We define the following classification:[21]

- No preference: $\alpha_1$ and $\alpha_2$ are not jointly significant.
- Preference for variety: $\alpha_1 < 0$, $\alpha_2 > 0$ and $\alpha_1 + \alpha_2 = 0$.
- Preference for boys: $\alpha_1 + \alpha_2 < 0$.
- Preference for girls: $\alpha_1 + \alpha_2 > 0$.

### 3.4.3 Identification assumptions

The main threat to identification is the prevalence of child mortality. In our sample, 15.7% of children died before turning five years old. In this context, when we analyse fertility choices, shall we consider the gender composition of the previous births or the gender composition of children alive at the time of the decision? There is a trade-off between exogeneity and relevance. The composition that matters to parents is probably among children who survived; but it is correlated to parents' choices regarding breastfeeding, nutrition and caring. Indeed, the proportion of sons among survivors could be an outcome of parents' gender preferences. That is why we consider the proportion among all births. Our strategy is close to an instrumental variable framework: we use the composition among births as an instrument for the composition among survivors, and estimate the reduced form.[22]

The first key identification assumption is that there is no sex-selective abortion. We believe that it is likely to hold because sex ratio at birth in our sample is equal to 51.2%, which is the ratio observed in Western countries (Brian and Jaisson (2007), Ben-Porath and Welch (1976) in the US, Jacobsen, Moller, and Mouritsen (1999) in Denmark) and generally considered as the natural level. Moreover, abortions are rare in Africa. Abortion is allowed without restriction only in Tunisia and in South Africa (United Nations, 2011b). According to recent estimations including illegal abortions, the number of abortions per 100 live births is 17 in Africa, compared to 34 in Asia and 59 in Europe (Sedgh, Henshaw, Singh, Ahman, and Shah, 2007). Last, sex-selective abortions are even less likely in our context, as obstetric ultrasound is not so common. Today, only 30% of women in cities, and 6% of women in rural areas have access to ultrasound during their pregnancy in Sub-Saharan Africa (Carrera, 2011).

The second identification assumption is that there is no sex-selective child mortality.[23] Otherwise, the coefficients in the reduced form capture both the reaction to the death of a child

[21] We do not consider the case $\alpha_1 > 0$, $\alpha_2 < 0$ and $\alpha_1 + \alpha_2 = 0$ because we never observe it in our estimations.

[22] Note that we cannot apply a 2SLS procedure because the outcome does not depend linearly on the instrumented variable. As a robustness check, we do the same analysis using the fraction of sons among survivors at the time of conception. We compare the indicator $\alpha_1 + \alpha_2$ obtained from this analysis to our main indicator, and we find a very high correlation between the two (0.97). Our conclusion regarding spatial heterogeneity is unchanged.

[23] Sex-selective adult mortality could also bias our estimates if parents form beliefs about the survival probability of their sons and daughters at adult age, and take fertility decisions according to these beliefs. However, qualitative evidence provided by demographers do not support the idea that people make such calculations about child loss (Randall and LeGrand, 2003).

and the "true" impact of gender composition on the next birth. In our sample, boys tend to die more than girls: for 100 girls dying before age five, 111 under-five boys die. This figure is 112 in Sub-Saharan Africa, 105 in North Africa, and it is above 100 in every country.[24] Consequently, families with more sons at birth are more likely to have lost one child. If parents intensify fertility after the death of a child, we would observe that families with more sons have shorter birth intervals. So we might tend to underestimate son preference and to overestimate daughter preference everywhere.[25] Another question is whether the variation across countries arises mainly from the variation in sex-selective mortality. In Section 3.6.1, we show that our findings are robust to a potential mortality bias by focusing on parents who lost no child.

Another threat to our strategy is that the prevalence of maternal mortality could lead to sample selection. In particular, if mothers exhibiting specific gender preferences are more likely to die, surviving women would be selected. For instance, suppose that mothers with the strongest son preference shorten birth intervals when they have only girls, increasing their exposure to maternal mortality risk. These women would be under-represented, and our estimate of son preference for surviving mothers would underestimate son preference in the whole population. Conversely, in a daughter preference setting, we would underestimate daughter preference. So selective maternal mortality might lead to underestimating gender preferences.[26] In our data, we can get indirect evidence of selective maternal mortality by looking at the sex ratio of the first born child. In our sample, the ratio is around $0.51$ in all countries, but we do find that in some countries, it increases (up to 0.57 in Nigeria), or decreases (down to 0.45 in Sudan) for older women. This is a hint that maternal mortality may be linked to gender preferences in different ways for different countries. We estimate an order of magnitude of the selection bias in Section 3.6.2 and find that it is too small to change qualitatively our findings.

## 3.5 Results

### 3.5.1 Comparative descriptive analysis

#### Heterogeneity over space

Figure 3.3 maps the magnitude of our indicator of gender preferences $(\alpha_1 + \alpha_2)$ for countries in which $\alpha_1$ and $\alpha_2$ are jointly significant. Otherwise, countries are classified as "no preference".[27] We find evidence of son preference in North Africa (Morocco, Tunisia, Egypt), Mali

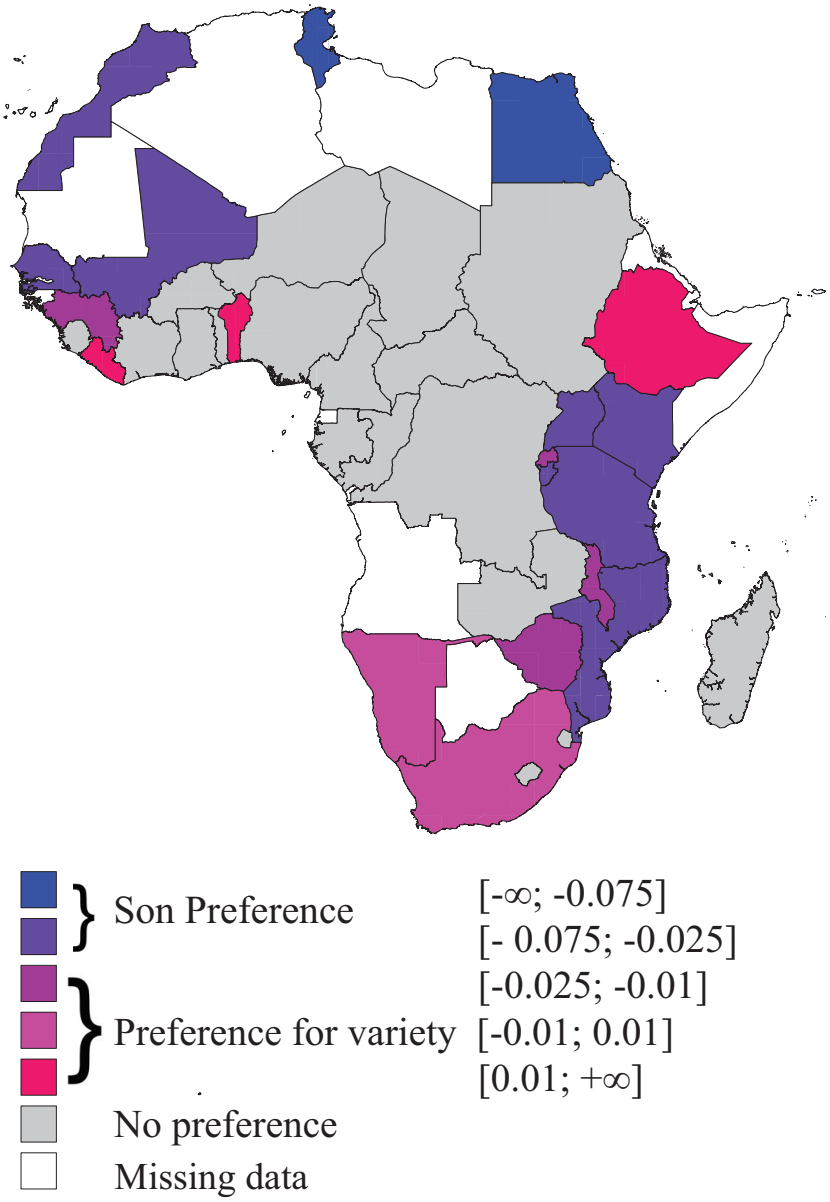
[24] Such a female advantage in mortality is observed in most countries in the world, with the notable exception of India and China. The average world ratio excluding India and China was around 111 during the last decades (United Nations, 2011a).

[25] Since differential mortality is the lowest in North Africa, it is the region in which son preference would be less underestimated.

[26] Note that another mechanism could lead to the same bias: if mothers having preferences for sons are more likely to *forget* first-born girls who died in their first days of life than first-born boys, this sex-selective recall would also lead to an underestimation of gender preferences.

[27] The classification of countries can also be found in Appendix D, in Table A-3.6.

**Figure 3.3:** Gender preferences by country: magnitude of the indicator $(\alpha_1 + \alpha_2)$




The map relates to our classification in the following way:

- No preference: $\alpha_1$ and $\alpha_2$ are not jointly significant.
- Son preference: $\alpha_1 + \alpha_2$ significantly negative.
- Preference for variety: $\alpha_1 + \alpha_2$ not significantly different from zero.
- Daughter preference (empty category): $\alpha_1 + \alpha_2$ significantly positive.

and Senegal, and also in the Great Lakes region (Burundi, Kenya, Uganda, Mozambique, Tanzania). Then, Southern Africa (Namibia and South Africa) is characterized by a preference for variety, and Central Africa (Cameroon, Chad, Congo, Congo DRC, Gabon, Central African Republic) by the absence of revealed gender preferences. In the rest of the continent, countries are divided into no gender preferences (Swaziland, Nigeria, Sierra Leone, Burkina Faso, Ghana, Togo, Niger, Sudan, Zambia, Lesotho and Madagascar, Cote d'Ivoire) and a taste for balance (Guinea, Liberia, Benin, Ethiopia, Rwanda, Malawi and Zimbabwe). No country displays daughter preference.

Such a simple sorting fails to give a sense of magnitude. To estimate by how much gender preferences impact fertility choices, we compute the predicted median birth spacing and the probability of short birth spacing ($\leq 24$ months) [28] for (i) couples with no son; (ii) couples with no daughter; and (iii) couples having the optimal mix of sons and daughters.[29] Estimations are reported in Table 3.1 for countries in which we found evidence of gender preferences; examining the magnitude is indeed meaningless when the proportion of sons has no significant impact on subsequent births.

North African countries stand out because they display the largest magnitudes. For instance, in Egypt, having no son is predicted to reduce the median birth spacing by three months as compared to having no daughter, and by five months as compared to the optimal mix of sons and daughters. Having no daughter (respectively having the optimal mix) decreases by 13% (respectively by 20%) the probability to have short birth intervals, compared to having no son. There, son preference has a strong impact on fertility patterns. Large magnitudes are also observed in South Africa. When the gender composition is perfectly balanced, couples are predicted to wait seven months more than couples having only boys or only girls. The taste for balance therefore translates into sizeable differences between families. In the rest of Africa, gender preferences have a much weaker impact. Should they display preferences for boys or for variety, all countries exhibit very small differences in predicted birth spacing across our three categories of interest (zero or one month). In case of son preference, having only sons decreases by roughly 4% the probability of short birth spacing as compared to having only daughters.

[28] In the Cox model, one can derive an estimate of the survival function $\widehat{S}(t)$ (Box-Steffensmeier and Jones, 2004). The predicted median birth spacing is $\tau$ s.t. $\widehat{S}(\tau) = 0.5$ and the probability of short birth spacing is $\widehat{S}(24)$.

[29] The optimal mix is the fraction of sons corresponding to the lowest hazard rate; it is equal to $-\frac{\alpha_1}{2\alpha_2}$.

**Table 3.1:** Magnitude of gender preferences across countries

| | Predicted median birth spacing (in months) | | | Probability of short intervals ($\leq 24$ months) | | |
|---|---|---|---|---|---|---|
| | No son | No daughter | Optimal mix | No son | No daughter | Optimal mix |
| **Countries displaying son preference** | | | | | | |
| Egypt | 29 | 32 | 34 | 35.4% | 30.9% | 28.5% |
| Tunisia | 27 | 28 | 31 | 39.5% | 36.5% | 32.5% |
| Burundi | 30 | 30 | 30 | 28.7% | 27.1% | 27.1% |
| Kenya | 28 | 29 | 29 | 33.6% | 32.1% | 31.4% |
| Uganda | 26 | 27 | 27 | 37.8% | 36.2% | 36.0% |
| Mali | 27 | 27 | 28 | 35.9% | 34.5% | 33.8% |
| Mozambique | 33 | 33 | 33 | 23.7% | 22.7% | 22.5% |
| Morocco | 24 | 25 | 26 | 46.6% | 45.0% | 41.8% |
| Tanzania | 33 | 33 | 33 | 22.5% | 21.8% | 21.4% |
| Senegal | 30 | 31 | 31 | 27.7% | 26.8% | 26.8% |
| **Countries displaying preferences for variety** | | | | | | |
| Zimbabwe | 35 | 36 | 36 | 18.8% | 18.4% | 17.8% |
| Rwanda | 28 | 28 | 29 | 34.0% | 33.4% | 32.1% |
| Guinea | 33 | 33 | 34 | 22.9% | 22.6% | 20.7% |
| Malawi | 33 | 33 | 34 | 22.1% | 21.9% | 21.3% |
| Namibia | 35 | 35 | 36 | 23.1% | 23.0% | 21.9% |
| South Africa | 58 | 58 | 65 | 13.5% | 13.6% | 12.2% |
| Benin | 33 | 33 | 34 | 22.0% | 22.3% | 20.9% |
| Ethiopia | 30 | 30 | 31 | 29.4% | 29.7% | 28.7% |
| Liberia | 26 | 25 | 26 | 41.6% | 42.4% | 39.9% |

We use the survival function and the parameters estimated in the Cox model. The optimal mix is the proportion of sons corresponding to the lowest predicted hazard rate; it is equal to $-\frac{\alpha_1}{2\alpha_2}$. The predicted median birth spacing and the probability of short birth spacing are computed for the median individual in each country. We sort the countries according to the magnitude of our indicator of gender preferences $(\alpha_1 + \alpha_2)$ (cf. Table A-3.6 in Appendix). We do not provide estimations for countries displaying no gender preferences, because the question of the magnitude makes no sense in this case.

**Heterogeneity over time**

To study the evolution of gender preferences over time, we interact our variables of interest with the mother's birth cohort in the general model:

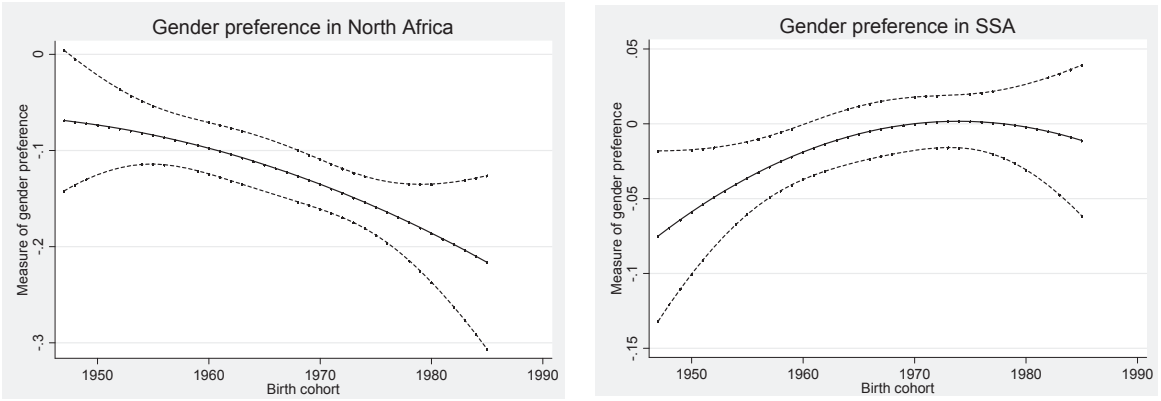
$$\begin{aligned}\lambda(t) &= \lambda_0(t) \times \exp(\lambda_1.cohort + \lambda_2.cohort^2 + \zeta_1.Frac_n + \zeta_2.Frac_n^2 + \phi_1.cohort.Frac_n + \\ &\phi_2.cohort^2.Frac_n + \omega_1.cohort.Frac_n^2 + \omega_2.cohort^2.Frac_n^2 + \theta.X_n + \kappa.C)\end{aligned}$$

We pool all the surveys together, adding a vector of dummies for each country ($C$) to our main specification. From the estimates, we compute an indicator of cohort-by-cohort gender preference:

$$\widehat{Pref} = \widehat{\zeta_1} + \widehat{\zeta_2} + \widehat{\phi_1}.cohort + \widehat{\phi_2}.cohort^2 + \widehat{\omega_1}.cohort + \widehat{\omega_2}.cohort^2$$

Considering birth spacing rather than completed fertility allows us to look at contemporary cohorts, because we already have information about the behavior of women born in the 70s and 80s.

**Figure 3.4:** The evolution of $(\alpha_1 + \alpha_2)$ over time




The graphs plot an indicator of cohort-by-cohort gender preferences, using an order 2 polynomial of mother's birth cohort. The dotted lines represent the 5% confidence intervals. Cox estimation. Standard errors clustered at the mother level. Weights. Controls: country, rank of preceding birth, and mother's characteristics: age at preceding birth, religion, family system, union type, education, wealth, area of residence, employment status. Beware that the scale is not the same in the two graphs.

In Figure 3.4, we plot $\widehat{Pref}$ against the birth cohort, together with its 5% confidence intervals, in North Africa and in Sub-Saharan Africa. Our indicator is more and more negative over time in North Africa, meaning that son preference increases. When we break down this trend by country, we find that it is mainly driven by Egypt, especially in the most recent years. As explained in Section 3.3.1, we have few mothers born after 1970 in Morocco and Tunisia. Before that date, the trend observed in Morocco is similar to the Egyptian one, whereas it is rather flat in Tunisia – if anything, son preference tends to decrease. The reinforcement of son prefer-

ence in North Africa coincides with fertility transitions, supporting the idea that a reduction in family size exacerbates gender preferences.

In Sub-Saharan Africa, there is not much variation: our indicator is fairly stable across the period. It is slightly increasing during the first decades, but the magnitude of the change is small and imprecisely estimated. For most cohorts, $\widehat{Pref}$ is not significantly different from zero, so there is no evidence of son preference.

### 3.5.2 Key drivers of gender preferences

Now that we have underscored different patterns in Sub-Saharan and in North Africa, one may wonder if socioeconomic factors drive some heterogeneity within these areas. In this section, we examine whether preferences vary across categories of women (e.g. educated vs. non-educated, rural vs. urban, matrilineal vs. patrilineal etc.).

#### Empirical strategy

We introduce interaction terms in our specification:

$$\lambda(t) = \lambda_0(t) \times \exp(\gamma_0.Alter + \gamma_1.Frac_n + \gamma_2.Frac_n^2 + \delta_1.Frac_n.Alter + \delta_2.Frac_n^2.Alter + \theta.X_n + \kappa.C)$$

Where $Alter$ is a dummy equal to 0 if women belong to the reference category, and 1 if they belong to the alternative category. Again, we pool all the surveys together, and we add country and cohort dummies. Then, for each category, we compute our indicator of gender preference (using the notations of the general specification, it corresponds to $\alpha_1 + \alpha_2$):

- Reference category: we test if $\gamma_1 + \gamma_2 = 0$
- Alternative category: we test if $\gamma_1 + \gamma_2 + \delta_1 + \delta_2 = 0$

We can conclude that gender preferences are different between the two categories if $\delta_1 + \delta_2$ is significantly different from 0.

#### Are religion and family systems shaping preferences?

We start by considering two structural factors that appeared in the literature review as potential drivers of gender preferences: Islamic influence and traditional kinship structure. We can only perform this analysis in Sub-Saharan Africa, because the number of Christians and matrilineal group members is too low in North Africa.

The first hypothesis we want to test is whether son preference is stronger among Muslims. The depth of Islamic penetration is indeed one way to explain the difference between North and Sub-Saharan African countries. The next question is whether, in Sub-Saharan Africa, Muslims and other religious groups have different preferences. In the first column of Table 3.2, we show that Muslims exhibit the same taste for balance as Christians and animists. Coefficients on the interaction terms are not significant, and the indicator of gender preferences is the same in both categories. So religion may play a role at the macro level, by influencing family law, property rights, social norms etc., but in a given institutional setting, it does not seem to drive individual gender preferences.

**Table 3.2:** Testing the impact of religion and family system in Sub-Saharan Africa

| Reference category<br>Alternative category | Christians and animists<br>Muslims | Patrilineal<br>Matrilineal | Monogamous<br>Polygamous |
|---|---|---|---|
| $frac(\gamma_1)$ | -0.131*** | -0.161*** | -0.210*** |
| | (0.031) | (0.026) | (0.031) |
| $frac^2(\gamma_2)$ | 0.119*** | 0.150*** | 0.197*** |
| | (0.030) | (0.025) | (0.030) |
| $frac \times Alter(\delta_1)$ | -0.041 | 0.248*** | 0.253*** |
| | (0.044) | (0.059) | (0.052) |
| $frac^2 \times Alter(\delta_2)$ | 0.048 | -0.216*** | -0.242*** |
| | (0.041) | (0.056) | (0.049) |
| $(\gamma_1 + \gamma_2)$ | -0.013 | -0.011* | -0.013 |
| $(\gamma_1 + \gamma_2 + \delta_1 + \delta_2)$ | -0.006 | 0.021 | 0.001 |
| Pvalue test $(\delta_1 + \delta_2) = 0$ | 0.65 | 0.10 | 0.51 |
| Observations | 2615663 | 2718999 | 2283793 |

Dependent variable: duration between births $n$ and $(n+1)$. $frac$: proportion of boys among the previous $n$ children. $Alter$: dummy for alternative category. Cox estimation, no hazard ratio. Standard errors clustered at the mother level. ***Significant at the 1 percent level. **Significant at the 5 percent level. *Significant at the 10 percent level. Weights. Controls: country, rank of preceding birth, and mother's characteristics: birth cohort, age at preceding birth, religion, family system, union type, education, wealth, area of residence, employment status, contraceptive use.

The second hypothesis is that son preference would prevail in patrilineal ethnic groups, while daughter preference should be observed in matrilineal groups. As shown in Table 3.2, column 2, this hypothesis is validated: we find that our indicator of gender preferences is negative in patrilineal groups, which means son preference; whereas it is positive in matrilineal groups, although not significant. Given the small proportion of the sample belonging to a matrilineal group (5.7%), we lack some power to take a definitive stance, but matrilineal groups seem to exhibit daughter preference. In any case, their preferences are significantly different from the ones in patrilineal groups. When we further split the sample on the median wealth index, we find that our result on kinship structure is driven by relatively rich people. In the poorest half of the sample, couples in patrilineal and matrilineal groups exhibit the same preferences. One interpretation might be that inheritance rules impact gender preferences only when families have enough assets to bequest.

In the third column of Table 3.2, we test if gender preferences differ across union types: polygamous vs. monogamous. We find preference for variety in monogamous unions, but no evidence of gender preferences in polygamous unions. For them, the coefficients on $Frac$ and $Frac^2$ are very close to zero. Given the caveat that we mentioned in the introduction, this result is not surprising: our unitary model is better-suited to monogamous households than to more complex household structures.

**How do preferences relate to modernization?**

Now, we turn to the modernization hypothesis, and examine individual indicators of development. Table 3.3 reports the results for Sub-Saharan Africa in the upper part, and for North Africa in the lower part. More precisely, we compare the poorest half and the richest half of the sample (column 1); non-working and working women (column 2); non-educated and educated women (column 3); rural and urban women (column 4); each time controlling for all other socioeconomic variables.

**Table 3.3:** Testing the impact of modernization factors

| Reference category<br>Alternative category | Poorest half<br>Richest half | No education<br>Education | Not working<br>Working | Rural<br>Urban |
|---|---|---|---|---|
| Panel A: Sub-Saharan Africa | | | | |
| $frac(\gamma_1)$ | -0.080** | -0.112*** | -0.085** | -0.159*** |
| | (0.032) | (0.033) | (0.033) | (0.029) |
| $frac^2(\gamma_2)$ | 0.072** | 0.105*** | 0.065** | 0.148*** |
| | (0.030) | (0.031) | (0.032) | (0.027) |
| $frac \times Alter(\delta_1)$ | -0.243*** | -0.083** | -0.129*** | 0.024 |
| | (0.047) | (0.042) | (0.041) | (0.047) |
| $frac^2 \times Alter(\delta_2)$ | 0.239*** | 0.078** | 0.145*** | -0.023 |
| | (0.044) | (0.039) | (0.038) | (0.044) |
| $(\gamma_1 + \gamma_2)$ | -0.008 | -0.007 | -0.019** | -0.010 |
| $(\gamma_1 + \gamma_2 + \delta_1 + \delta_2)$ | -0.011 | -0.012 | - 0.003 | -0.010 |
| Pvalue test $(\delta_1 + \delta_2) = 0$ | 0.80 | 0.74 | 0.22 | 0.96 |
| Observations | 2446170 | 2717402 | 2716216 | 2718999 |
| Panel B: North Africa | | | | |
| $frac(\gamma_1)$ | -0.010 | -0.157*** | -0.681*** | -0.435*** |
| | (0.068) | (0.043) | (0.035) | (0.042) |
| $frac^2(\gamma_2)$ | -0.128** | 0.027 | 0.541*** | 0.281*** |
| | (0.063) | (0.040) | (0.033) | (0.039) |
| $frac \times Alter(\delta_1)$ | -1.523*** | -1.514*** | -1.082*** | -0.796*** |
| | (0.079) | (0.057) | (0.081) | (0.057) |
| $frac^2 \times Alter(\delta_2)$ | 1.482*** | 1.488*** | 1.072*** | 0.825*** |
| | (0.073) | (0.053) | (0.075) | (0.053) |
| $(\gamma_1 + \gamma_2)$ | -0.137*** | -0.130*** | -0.140*** | -0.154*** |
| $(\gamma_1 + \gamma_2 + \delta_1 + \delta_2)$ | -0.179*** | -0.155*** | - 0.149*** | -0.126*** |
| Pvalue test $(\delta_1 + \delta_2) = 0$ | 0.10 | 0.15 | 0.69 | 0.11 |
| Observations | 209775 | 385033 | 385177 | 385180 |

Dependent variable: duration between births $n$ and $(n+1)$. $frac$: proportion of boys among the previous $n$ children. $Alter$: dummy for alternative category. Cox estimation, no hazard ratio. Standard errors clustered at the mother level. ***Significant at the 1 percent level. **Significant at the 5 percent level. *Significant at the 10 percent level. Weights. Controls: country, rank of preceding birth, and mother's characteristics: birth cohort, age at preceding birth, religion, family system, union type, education, wealth, area of residence, employment status, contraceptive use.
0.8

Starting with Sub-Saharan Africa, we first find that wealth is correlated with the intensity of gender preferences, but not with the preferred proportion of sons. In the richest half of the sample, coefficients on $Frac$ and $Frac^2$ are both significantly larger in absolute terms than in the poorest half; but the indicator of preferences for boys vs. girls is the same and reveals preferences for variety. Wealth seems to modify the *magnitude*, but not the *nature* of gender preferences. While the same findings hold, to a smaller extent, for education, we find no correlation between gender preferences and the area of residence controlling for education, wealth, and employment status.

The second result is that son preference is very strong for women who do not work, whereas working mothers exhibit preferences for variety. One explanation emphasizes insurance motives for the mother: non-working women are heavily dependent on their husband, and in case of widowhood, on their sons. The prevalence of son preference among non-working mothers also holds in matrilineal groups, suggesting that the insurance motive might prevail over the lineage motive.

Turning to North Africa, we find again that the magnitude of preferences is much larger in more "modern" categories. But the nature of preferences is not substantially affected: there is evidence of a strong son preference in every category we consider.

### 3.5.3 Mechanisms: individual choices or social norms?

In this section, we discuss how gender preferences may translate into differential spacing behavior. The first channel we have in mind is a conscious choice made by the couple to bring forward or to delay the next birth in order to reach an ideal gender composition. This requires that couples have some control over birth spacing, be it through modern contraceptives or more traditional methods such as varying breastfeeding duration. Another channel deserves attention: there might be social norms surrounding birth spacing practices that produce differential spacing. We think in particular of breastfeeding norms that require children of one gender to be fed longer than others, revealing gender preferences at the society level.[30]

In Sub-Saharan Africa, breastfeeding practices do not differ by gender, as already mentioned in the literature review (Chakravarty, 2012; Garenne, 2003). The equal treatment in breastfeeding might reflect the absence of gender preferences at both individual and social levels. Regarding the modern contraceptive channel, it affects a limited fraction of the population. According to the United Nations, the modern contraception prevalence in Sub-Saharan Africa was 20% in 2011. As a comparison, the same figure in Asia was 61% (United Nations, 2012). Interestingly, those women who use modern contraceptives reveal stronger preferences for variety than non-users.[31] This result points to a deliberate manipulation of birth intervals.

In North Africa, a larger share of the population uses modern contraceptive methods: the contraception prevalence was 48% in 2011 (United Nations, 2012). As already shown in earlier studies (Yount *et al.*, 2000), contraceptive use depends on the gender composition of earlier births. In our sample, the proportion of women using modern birth control methods is significantly lower (by 1.7 percentage

[30] Since breastfeeding duration is correlated to the duration of postpartum insusceptibility, such norms may generate systematic differences in birth intervals after the birth of a son vs. a girl (Jayachandran and Kuziemko, 2011).

[31] If we run the same regression as in section 3.5.2, coefficients on $Frac$ and $Frac^2$ are larger in absolute terms for users than for non-users. In both categories, the indicator of gender preferences is not significantly different from zero.

points) after the birth of a girl as compared to a boy. Therefore, the shorter birth intervals observed in families with more daughters are partly generated by different contraceptive choices. By comparing users and non-users, we find again that contraceptive use heightens the magnitude of preferences, but does not affect the nature of preferences being revealed. This is a first hint that our strategy captures intentional choices. As for breastfeeding practices, they differ by gender only in Egypt, where we find that boys are significantly more likely than girls to be breastfed longer than 15 months (by two percentage points). This result is in line with Chakravarty (2012) and may originate from individual choices as well as from social norms. We attempt to disentangle both dimensions by decomposing our variable $Frac_n$ into $Frac_{n-1}$: the proportion of sons among the previous $(n-1)$ births, and $Boy_n$: a dummy indicating whether the n$^{th}$ birth is a boy. Results for North Africa are reported in Table A-3.7 in Appendix E. The proportion of sons among the previous $(n-1)$ births has an impact on the duration between births $n$ and $(n+1)$: we find the same asymmetric U-shape as with $Frac_n$. It indicates that parents take into account the gender composition of their whole family. Parents also react to the gender of the latest born: the coefficient on $Boys_n$ is negative and significant, which may reflect conscious or less conscious choices. One argument in favor of conscious choices is that the impact of $Boys_n$ differs across birth orders: it is stronger at ranks three and four than at other ranks. Such a pattern is difficult to reconcile with a breastfeeding norm.

All in all, we believe that differential spacing should be mainly interpreted as a reflection of conscious, individual choices. Social norms surrounding breastfeeding may also play a role, but the body of evidence seems to indicate that it is limited.

## 3.6 Robustness Tests

### 3.6.1 Testing the child mortality bias

As mentioned in Section 3.4.3, we test if sex-selective mortality introduces a bias in our estimates. The idea is to isolate couples who lost at least one child among the previous $n$ births, and to focus on couples who lost no child. If we find evidence of gender preferences for the latter, they cannot be driven by differential mortality, they have to be driven by differential fertility rules. In Table A-3.8 in Appendix E, we interact $Frac$ and $Frac^2$ with a dummy equal to one if at least one of the previous $n$ children died. As expected, we find that the sex-selective mortality leads us to underestimate the extent of son preference in Africa: our indicator $(\alpha_1 + \alpha_2)$ is more negative among women who did not lose any children than in the baseline. However, the difference is small, around 10%. Then, we check if our baseline classification of countries into the "son preference group" and the "preference for variety group" remains valid. In Table A-3.9, in Appendix E, we find again that focusing on couples who lost no child slightly shifts our results towards more son preference. However, $(\alpha_1 + \alpha_2)$ remains not significantly different from zero in the group classified as "preferences for variety", implying that our findings are robust.

To better understand the magnitude of the bias, we compute the predicted median birth spacing and the probability of stopping for couples who did not lose a child. In absolute values, birth spacing increases by one month, and the probability of stopping by roughly one percentage point, compared to

the magnitudes discussed in section 3.5.1. But in relative terms, the differences across categories remain very stable.

The last test is to look how our sorting of countries is affected by mortality. Going on with the same specification, we compute $(\alpha_1 + \alpha_2)$ among people who lost no child in each country, and compare it to our baseline $(\alpha_1 + \alpha_2)$. The correlation between both indicators is 0.97. Then, we sort the countries according to each indicator, and the rank correlation is equal to 0.95. In the end, the variation in sex-selective mortality across countries does not seem to drive the variation we observe.[32]

### 3.6.2 Testing the sample selection of mothers

We further deal with a potential selection bias. We run our model on mothers below 40 years old who are less likely to have died or forgotten their earlier-born children.[33] For this sample of younger mothers, we find that the gender of the first born is exogenous to socioeconomic characteristics, which supports the assumption that the sample is not selected. The correlation between the indicator computed on younger mothers and our baseline indicator is $0.94$. Furthermore, the rank correlation between both classifications is $0.88$. These strong correlations supports the idea that our results are not driven by differences between countries in maternal mortality or recall bias. Last, we checked, as in Table A-3.9, that our classification of countries into "preference for son" and "preference for variety" remains robust once we restrict our sample to younger mothers.

One limit of this strategy is that maternal mortality affects also young women. In particular, one can fear that when we find no gender preferences in some countries, it might be due to the fact that women having stronger son preference massively died before 40 years old. Let us consider simple back of the envelope calculations to get an order of magnitude of such a downward bias. In countries classified as "no preference", let us assume that there are in fact two groups of women. The first one, accounting for a proportion $M$ of the population, has very strong son preference; we attribute to them the largest magnitude found in our sample ($\alpha_1 + \alpha_2 = -0.17$ in Egypt). The second group has no gender preference; for them, $\alpha_1 = \alpha_2 = 0$. The scenario that would lead to the most extreme selection bias is that all women having a son preference die, while all women having no preference survive. In this case, $M$ represents the mortality rate. Following our baseline strategy, we would only observe surviving women and compute an indicator of gender preferences equal to 0, whereas the true indicator for the whole population would be around $-0.17 \times M$.[34] These countries could therefore reach the lowest magnitude of son preference observed in our sample ($\alpha_1 + \alpha_2 = -0.04$ in Senegal) if they had a maternal mortality rate at least equal to $M = \frac{0.04}{0.17} = 23.5\%$. It amounts to a lifetime risk of one out of four, a magnitude never reached in Sub-Saharan Africa.[35] To conclude, maternal mortality would need to reach unlikely high levels in order to drive our "no preference" results.

[32] Another piece of evidence is given by the low rank correlation (0.16) between our sorting and the sorting of countries on the child mortality ratio between girls and boys.

[33] Results not shown, but available on request.

[34] Under the technical assumption that the weighted average of our indicator in both groups is a good proxy for the global indicator.

[35] By comparison, it is almost ten times higher than the average risk in Sub-Saharan Africa in 2013 (one out of 38), and four times larger than the largest risk (one out of 15 in Chad) (WHO, UNICEF, UNFPA and The World Bank, 2014).

### 3.6.3 Investigating heterogenous effects across birth ranks

In our model, we pooled all birth ranks together to have more power and to build a single indicator of gender preferences. But some papers in the literature on India have discussed the heterogeneous effect of gender preferences across birth ranks. For instance, Jayachandran and Kuziemko (2011) show that the impact of son preference is stronger at higher ranks, when parents get closer to their ideal family size. On the other hand, family size itself may influence preferences: Jayachandran (2014) finds that son preference increases when families get smaller.

Our strategy might easily be modified to investigate whether the impact of gender preferences differs across birth ranks in Africa.[36] We estimate a model similar to Section 3.5.1: we interact our variables of interest with the child's birth rank, and we compute a rank-by-rank indicator. In Figure A-3.1, in Appendix E, we plot this indicator together with its 5% confidence intervals in North Africa and in Sub-Saharan Africa. Both graphs display a U-shape, meaning that son preference is the strongest between ranks three and six. At lower and higher ranks, it is much weaker in North Africa, and disappears in Sub-Saharan Africa.

There are three ways to explain this pattern. First, the intensity of gender preferences, for the same couple, may vary across birth orders. It could be low at lowest ranks, because parents still have time for other tries in the future. Then the intensity could increase as time passes by, and parents start worrying about the eventual gender composition of their children. Last, the trend could revert at highest ranks if parents can already count on their eldest children. Second, the ability to control fertility, and hence to translate preferences into differential spacing, may be higher at intermediary ranks. Indeed, at lowest ranks, contraceptive use is less widespread; and at highest ranks, biological fecundity is lower, which reduces women's leeway. The last explanation is that gender preferences may be heterogenous across couples depending on the family size. Such a U-shape is consistent with (i) preference for variety in small size families, (ii) son preference in middle size families, and (iii) no preference in large size families.[37]

These results raise some concern about the implicit assumption in our model that birth orders have a multiplicative effect on the baseline hazard. To remove any doubt, we estimated our model rank by rank, for each country. We retrieved coefficients $\alpha_1$ and $\alpha_2$ for each rank, and we computed a weighted average, taking into account the number of observations at each rank. We were thus able to build a new indicator of gender preferences for each country, and we checked that it was strongly correlated to our baseline indicator (0.86). The restriction we made on birth order effects in our main specification does not change qualitatively our results.

[36] In Appendix F, we focus on the situation after the second birth.

[37] When we interact $Frac$ and $Frac^2$ with family size instead of birth order (using the sub-sample of women over 40 years old), we also find a U-shape, which provides support for this explanation.

## 3.7 Conclusion

All in all, we find robust evidence that son preference influences fertility patterns in North Africa. Indeed, people tend to shorten birth spacing and to have additional children as long as they have not had enough sons. This has strong implications for gender inequality: an average girl would be weaned sooner, and would face more competition from her siblings, than an average boy. Moreover, women, as mothers, would put their own lives in jeopardy to ensure that enough sons are born. Policies aiming at reducing women's reliance on sons, or equalizing the value of sons and daughters to their parents, could weaken the motives for son preference. Ultimately, they could help lengthening birth intervals and improving maternal and child health in North Africa.

We cannot draw the same conclusion for Sub-Saharan Africa: son preference exists in some countries, but it is weak. Overall, fertility behavior is rather consistent with a preference for variety or no preference at all. The impact of gender preferences on fertility patterns is not substantial enough to induce gender inequality. In this context, policies combating son preference will not be enough to curb fertility.

We also showed that differential spacing mainly reflects conscious choices, implemented through modern or traditional birth control methods, rather than social norms related to breastfeeding. In the likely scenario that some women still have unmet needs for family planning, our results suggest that gender preferences may deepen in the short run. The rise in contraceptive use might exacerbate son preference in North Africa and preference for variety in Sub-Saharan Africa, provided that current contraceptive users reveal preferences that are shared by the whole population. In the longer run, the evolution of gender preferences depends on more structural changes that could impact the nature of preferences, for instance through women empowerment.

The dissimilarity between North Africa and Sub-Saharan Africa can certainly be explained by structural differences in women's role in society. Yet, we are not claiming that gender inequality is only an issue in North Africa, but not in Sub-Saharan Africa. There are plenty of mechanisms by which gender preferences prevailing in a society may translate into inequality, beginning with family law and property rights. On many dimensions, it might be argued that women do have a subordinate status in Sub-Saharan Africa. Anderson and Ray (2010) show that, in this region, there are "missing women", too. But contrary to India and China, they are in majority of adult age; HIV/AIDS and maternal deaths are the two main sources of female excess mortality. The discrimination against women would appear later in life: at puberty? At marriage? At motherhood? At widowhood? Understanding when and why remains an open question.

# Appendix

## Appendix A: Additional information on data

**Table A-3.1:** Survey waves and years

| Country | Survey years | Nb births | Nb women |
|---|---|---|---|
| **Benin** | 1996, 2001, 2006, 2011 | 143,141 | 35,210 |
| **Burkina Faso** | 1992, 1998, 2003, 2010 | 140,428 | 32,396 |
| **Burundi** | 1987, 2010 | 36,406 | 8,731 |
| **Congo Dem. Rep.** | 2007 | 29,548 | 7,148 |
| **Cameroon** | 1991, 1998, 2004, 2011 | 98,566 | 25,266 |
| **Central African Rep.** | 1994 | 16,936 | 4,388 |
| **Chad** | 1996, 2004 | 47,187 | 10,508 |
| **Rep. of the Congo** | 2005, 2011 | 48,635 | 13,339 |
| **Cote d'Ivoire** | 1994, 1998, 2011 | 60,656 | 15,654 |
| **Egypt** | 1988, 1992, 1995, 2000, 2005, 2008 | 294,830 | 76,897 |
| **Ethiopia** | 2000, 2005 | 129,113 | 30,263 |
| **Gabon** | 2000, 2012 | 39,987 | 10,882 |
| **Ghana** | 1988, 1993, 1998, 2003, 2008 | 67,676 | 17,748 |
| **Guinea** | 1999, 2005, 2012 | 77,741 | 18,622 |
| **Kenya** | 1989, 1993, 1998, 2003, 2008 | 117,031 | 28,606 |
| **Lesotho** | 2004, 2009 | 29,137 | 10,023 |
| **Liberia** | 1986, 2007 | 39,387 | 9,932 |
| **Madagascar** | 1992, 1997, 2003, 2008 | 109,847 | 28,417 |
| **Malawi** | 1992, 2000, 2004, 2010 | 164,935 | 41,394 |
| **Mali** | 1987, 1995, 2001, 2006 | 150,720 | 32,570 |
| **Morocco** | 1987, 1992, 2003 | 80,669 | 18,970 |
| **Mozambique** | 1997, 2003, 2011 | 101,179 | 27,154 |
| **Namibia** | 1992, 2000, 2006 | 47,840 | 15,126 |
| **Niger** | 1992, 1998, 2006, 2012 | 131,290 | 27,403 |
| **Nigeria** | 1990, 1999, 2003, 2008 | 179,246 | 40,960 |
| **Rwanda** | 1992, 2005, 2010 | 82,151 | 19,838 |
| **Senegal** | 1986, 1992, 1997, 2005, 2011 | 144,101 | 33,956 |
| **Sierra Leone** | 2008 | 21,136 | 5,876 |
| **South Africa** | 1998 | 22,934 | 8,223 |
| **Sudan** | 1989 | 25,805 | 5,277 |
| **Swaziland** | 2006 | 11,410 | 3,488 |
| **Tanzania** | 1991, 1996, 1999, 2004, 2010 | 126,319 | 30,830 |
| **Togo** | 1988, 1998 | 37,051 | 8,825 |
| **Tunisia** | 1988 | 16,463 | 3,856 |
| **Uganda** | 1988, 1995, 2000, 2006, 2011 | 120,935 | 27,339 |
| **Zambia** | 1992, 1996, 2001, 2007 | 92,092 | 22,423 |
| **Zimbabwe** | 1988, 1994, 1999, 2005, 2010 | 82,134 | 24,606 |

The table lists all the DHS included in the analysis, reporting countries, years of survey as well as the number of births and women in each survey.

**Table A-3.2:** Matrilineal ethnic groups: matching the Ethnographic Atlas and DHS data

| Ethnic group in (Gray, 1998) | Country | Year of DHS | Code in DHS | Comments |
|---|---|---|---|---|
| Dorosie / Voltaic people | Burkina Faso | | | Ethnic group not listed in DHS |
| Lobi / Voltaic people | Burkina Faso | 1992, 1998, 2003, 2010 | 6 | |
| Tuareg | Burkina Faso | 1992, 1998, 2003, 2010 | 9 | |
| Udalan / Plateu Nigerians - Chadic | Burkina Faso | | | Ethnic group not listed in DHS |
| Fur / Darfur | Chad | | | Ethnic group not listed in DHS |
| Sundi / Central Bantu, basundi | Congo | 2005 | 9 | |
| Teke / Northwestern Bantu, bateke | Congo | 2005 | 22, 23 | |
| Teke / Northwestern Bantu, bateke | Congo | 2011 | 5 | |
| Yombe / Central Bantu, bayombe | Congo | 2005 | 12 | |
| Bembas (Luapula, Lamba, Buye, Kaonde, Lala) | DRC | | | Ethnicity variable too broad |
| Kasai (Bunda, Yanzi, Dzing, Sakata, Kuba, Lele) | DRC | | 7 | Ethnicity variable too broad |
| Kongos (Sundi, Yombe) | DRC | | | Ethnicity variable too broad |
| Kwango (Pende, Suku, Yaka) | DRC | | | Ethnicity variable too broad |
| Lunda (Luvale, Ndembu, Chokwe) | DRC | | 8 | Ethnicity variable too broad |
| Teke / Northwestern Bantu | DRC | | | Ethnicity variable too broad |
| Wodaabe, Bororos | DRC | | | Ethnicity variable too broad |
| Mpongwe / Northwestern Bantu | Gabon | | | Ethnic group not listed in DHS |
| Shogo / Northwestern Bantu | Gabon | 2000, 2012 | 6 | |
| Teke / Northwestern Bantu | Gabon | 2000, 2012 | 3 | |
| Twi Akan (Akyem, Anyi, Brong) | Ghana | 1988 | 1,2,3 | |
| Twi Akan (Akyem, Anyi, Brong) | Ghana | 1993 | 1,2,3,4 | |
| Twi Akan (Akyem, Anyi, Brong) | Ghana | 2003, 2008 | 1 | |
| Twi Lagoon (Assini) | Ghana | 1988 | 1,2,3 | |
| Voltaic people (Lobi, Kulango) | Ghana | | | Ethnic group not listed in DHS |
| Tenda | Guinea | | | Ethnic group not listed in DHS |
| Twi Akan (Baule, Anyi, Brong) | Cote d'Ivoire | 1998 | 1 | |
| Twi Akan (Baule, Anyi, Brong) | Cote d'Ivoire | 2011 | 101-106, 108-111, 114 | |
| Twi Lagoon (Avikam, Assini) | Cote d'Ivoire | | | Ethnic group not listed in DHS |
| Voltaic people (Lobi, Kulango) | Cote d'Ivoire | 1998 | 5 | Ethnicity variable missing in 1994 |
| Voltaic people (Lobi, Kulango) | Cote d'Ivoire | 2011 | 140, 146 | Ethnicity variable missing in 1994 |
| Chewas | Malawi | 2000, 2004, 2010 | 1 | |
| Nyanja / Maravi | Malawi | 2010 | 12 | |
| Nyasa / Maravi | Malawi | | | Ethnic group not listed in DHS |
| Yao | Malawi | 2000, 2004, 2010 | 5 | |
| Antessar / Tuareg | Mali | | | Ethnic group not listed in DHS |
| Udalan / Plateu Nigerians - Chadic | Mali | | | Ethnic group not listed in DHS |
| Chewas, chichewas | Mozambique | 2003 | 10 | |
| Chewas, chichewas | Mozambique | 1997 | 14 | |
| Chewas, chichewas | Mozambique | 2011 | 11 | |
| Kunda / Maravi | Mozambique | | | Ethnic group not listed in DHS |
| Makonde / Yao | Mozambique | 2011 | 15 | |
| Makonde / Yao | Mozambique | 1997 | 50 | |
| Nyanja / Maravi | Mozambique | 2003 | 26 | |
| Nyanja / Maravi | Mozambique | 1997 | 21 | |
| Nyasa / Maravi | Mozambique | | | Ethnic group not listed in DHS |
| Sena / Maravi, chisena | Mozambique | 2011 | 4 | |
| Sena / Maravi, chisena | Mozambique | 2003 | 5 | |
| Sena / Maravi, chisena | Mozambique | 1997 | 25 | |
| Yao | Mozambique | 2011 | 12 | |
| Ambo / Southwestern Bantu | Namibia | 1992 | 3 | |
| Ambo / Southwestern Bantu | Namibia | 2000 | 7 | |
| Ambo / Southwestern Bantu | Namibia | 2006 | 7 | |
| Tuareg (Azjer, Ahaggaren, Asben) | Niger | 1992, 1998 | 8 | Ethnicity variable missing in 2012 |
| Tuareg (Azjer, Ahaggaren, Asben) | Niger | 2006 | 7 | Ethnicity variable missing in 2012 |
| Udalan / Plateu Nigerians - Chadic | Niger | | | Ethnic group not listed in DHS |
| Daka / Eastern Nigritic | Nigeria | | | Ethnicity variable missing |
| Kurama, Gure /Bantoid People | Nigeria | | | Ethnicity variable missing |
| Longuda / Eastern Nigritic | Nigeria | | | Ethnicity variable missing |
| Ndoro /Bantoid People | Nigeria | | | Ethnicity variable missing |
| Tenda | Senegal | | | Ethnic group not listed in DHS |
| Sherbro / Ku and Peripheral Mande | Sierra Leone | 2008 | 16 | |
| Fur / Darfur | Sudan | | | Ethnicity variable missing |
| Nuba / Nubians (Midobi, Tumtum) | Sudan | | | Ethnicity variable missing |
| Makonde / Yao | Tanzania | | | Ethnicity variable missing |
| Nyasa / Maravi | Tanzania | | | Ethnicity variable missing |
| Ranji / Rift | Tanzania | | | Ethnicity variable missing |
| Sagara / Rufiji | Tanzania | | | Ethnicity variable missing |
| Zigula (Luguru, Nguru, Kwere) | Tanzania | | | Ethnicity variable missing |
| Bemba (Lamba, Lala, Kaonde, Luapula) | Zambia | 1996, 2001, 2007 | 1, 2, 3, 26, 27 | |
| Bemba (Lamba, Lala, Kaonde, Luapula) | Zambia | 1992 | 1 | |
| Lunda (Luvale, Ndembu, Luchazi) | Zambia | | | Ethnic group not listed in DHS |
| Maravi (Chewa, Kunda, Nyanja) | Zambia | 1996, 2001, 2007 | 48, 51, 52, 53 | |
| Maravi (Chewa, Kunda, Nyanja) | Zambia | 1992 | 5 | |
| Tonga / Middle Zambesi Bantu | Zambia | 1992 | 2 | |
| Tonga / Middle Zambesi Bantu | Zambia | 1996, 2001, 2007 | 19 | |
| Tonga / Middle Zambesi Bantu | Zimbabwe | | | Ethnicity variable missing |

The table shows how we matched ethnic groups coded by (Gray, 1998) to the ethnicity variable in DHS.

**Table A-3.3:** Number of children and birth spacing, by country

| Country | Number of children | Average birth intervals (in months) | Proportion of short intervals ($\leq 24$ months) |
|---|---|---|---|
| Benin | 6.3 | 35.5 | 26.7 |
| Burkina Faso | 7.0 | 34.8 | 25.4 |
| Burundi | 6.9 | 32.5 | 32.0 |
| CDR | 6.5 | 34.1 | 32.3 |
| Cameroon | 6.3 | 34.1 | 32.9 |
| Centrafrique | 6.2 | 32.9 | 35.2 |
| Chad | 7.2 | 31.2 | 35.2 |
| Congo-Brazza | 5.2 | 43.7 | 21.5 |
| Cote d'ivoire | 6.4 | 37.4 | 27.6 |
| Egypt | 5.3 | 33.3 | 40.3 |
| Ethiopia | 6.1 | 34.6 | 31.1 |
| Gabon | 5.5 | 41.1 | 29.9 |
| Ghana | 5.8 | 40.0 | 23.0 |
| Guinee | 6.4 | 37.0 | 23.5 |
| Kenya | 6.5 | 34.4 | 34.8 |
| Lesotho | 4.5 | 45.1 | 16.7 |
| Liberia | 6.2 | 37.2 | 32.2 |
| Madagascar | 6.2 | 33.8 | 38.3 |
| Malawi | 6.8 | 34.7 | 28.7 |
| Mali | 7.5 | 31.9 | 36.8 |
| Maroc | 6.2 | 33.7 | 40.1 |
| Mozambique | 6.0 | 36.3 | 28.2 |
| Namibia | 5.1 | 42.1 | 25.6 |
| Niger | 7.9 | 30.9 | 36.7 |
| Nigeria | 6.7 | 33.1 | 35.3 |
| Rwanda | 6.7 | 33.0 | 33.2 |
| Senegal | 6.7 | 34.3 | 30.6 |
| Sierra Leone | 5.6 | 38.6 | 28.1 |
| South Africa | 3.9 | 48.3 | 21.8 |
| Sudan | 7.6 | 29.1 | 45.5 |
| Swaziland | 5.5 | 41.7 | 26.2 |
| Tanzania | 6.6 | 36.1 | 26.8 |
| Togo | 6.7 | 35.9 | 24.8 |
| Tunisia | 6.3 | 29.4 | 46.9 |
| Uganda | 7.4 | 31.4 | 37.9 |
| Zambia | 7.1 | 34.4 | 28.2 |
| Zimbabwe | 5.6 | 40.7 | 21.3 |
| Total | 6.2 | 34.7 | 33.0 |

Weights. The number of children is computed on the sample of women over 40 years old

## Appendix B: Evidence from differential stopping rule

Can we infer the same type of gender preferences by examining only stopping rules, and not spacing rules? To avoid the issue of censoring, we restrict our sample to women over 40 years old, and we consider the following linear probability model:

$$Y_n = \alpha_0 + \alpha_1.Frac_n + \alpha_2.Frac_n^2 + \theta.X_n$$

Where $Y_n$ equals 0 when women stopped having children at parity $n$ and 1 when women gave birth to another child. $X_n$ remains the same vector of controls (a dummy for each rank and mother's characteristics). In Table A-3.4, we sort the countries according to the same criteria we used in the duration model. We find evidence of son preference in Egypt, Mozambique and Gabon, daughter preference in Sudan and preference for variety in Congo DRC, Guinea, Kenya, Morocco, Namibia and Ethiopia. But in the vast majority of countries, we cannot detect any differential stopping rule depending on the gender composition of previous children.

**Table A-3.4:** Ranking from differential stopping

| Country | $\alpha_1 + \alpha_2$ | pvalue of the sum | pvalue joint significance |
|---|---|---|---|
| **$\alpha_1$ and $\alpha_2$ are jointly significant** | | | |
| Gabon | -0.053 | 0.04 | 0.10 |
| Egypt | -0.035 | 0.00 | 0.00 |
| Mozambique | -0.032 | 0.08 | 0.06 |
| Congo DRC | -0.022 | 0.33 | 0.05 |
| Guinea | -0.017 | 0.15 | 0.00 |
| Kenya | -0.014 | 0.24 | 0.02 |
| Morocco | -0.009 | 0.41 | 0.00 |
| Namibia | 0.010 | 0.54 | 0.06 |
| Ethiopia | 0.010 | 0.27 | 0.02 |
| Sudan | 0.047 | 0.05 | 0.04 |
| **$\alpha_1$ and $\alpha_2$ are not jointly significant** | | | |
| Uganda | -0.023 | 0.07 | 0.19 |
| Tunisia | -0.020 | 0.36 | 0.36 |
| Tanzania | -0.019 | 0.15 | 0.22 |
| Cameroon | -0.019 | 0.17 | 0.17 |
| South Africa | -0.016 | 0.47 | 0.35 |
| Swaziland | -0.014 | 0.66 | 0.47 |
| Lesotho | -0.013 | 0.48 | 0.48 |
| Burundi | -0.012 | 0.47 | 0.53 |
| Zimbabwe | -0.011 | 0.38 | 0.12 |
| Togo | -0.007 | 0.68 | 0.74 |
| Congo | -0.006 | 0.79 | 0.94 |
| Rwanda | -0.006 | 0.54 | 0.62 |
| Madagascar | -0.006 | 0.66 | 0.80 |
| Burkina Faso | -0.005 | 0.62 | 0.84 |
| Sierra Leone | -0.004 | 0.87 | 0.46 |
| Nigeria | -0.002 | 0.85 | 0.97 |
| Mali | 0.000 | 0.97 | 0.97 |
| Niger | 0.000 | 1.00 | 0.99 |
| Chad | 0.000 | 0.98 | 1.00 |
| Central African Republic | 0.003 | 0.93 | 0.99 |
| Ghana | 0.008 | 0.52 | 0.72 |
| Benin | 0.008 | 0.37 | 0.62 |
| Zambia | 0.011 | 0.42 | 0.71 |
| Senegal | 0.011 | 0.33 | 0.36 |
| Malawi | 0.017 | 0.13 | 0.31 |
| Cote d'Ivoire | 0.031 | 0.14 | 0.28 |
| Liberia | 0.032 | 0.10 | 0.11 |

OLS estimation. Sample: women over 40 years old. Weights. Controls: country, rank of preceding birth, and mother's characteristics: birth cohort, age at preceding birth, religion, family system, union type, education, wealth, area of residence, employment status.

## Appendix C: At least one son or more sons?

One may wonder if son preference means that parents want many sons, or that they want at least one son. In the literature, this interpretation has been called the "funeral pyre" hypothesis, in reference to the Hindu tradition that requires a son for lighting the funeral pyre of the parents (Arnold *et al.*, 1998). Similarly, preferences for variety may refer to a preference for at least one child of each gender, or to a taste for a perfect balance in the gender composition of children. In a low fertility setting, both interpretations generally coincide. But in the African context, it is worth examining if there are gender preferences above and beyond "having at least one son and/or one daughter".

In Table A-3.5, we test if women having at least one child of each gender keep displaying gender preferences. The answer is positive: the quadratic relationship between the proportion of sons and the hazard rate still holds. Interestingly, coefficients are even larger in absolute values, meaning that the magnitude of gender preferences is stronger in the sub-population having at least one son and one daughter. A tentative explanation is that some people would believe that the probability to give birth to a boy vs. a girl might vary across couples, and they may try to infer their own probability from past outcomes. Couples having only sons or only daughters in the past may therefore believe that they would never have a child of the other sex, and delay the next try.

In the end, in such a high fertility setting, son preference means that parents want more sons than daughters; and preferences for variety mean that they want the same number of boys and girls.

**Table A-3.5:** Are there gender preferences beyond "at least one son / one daughter"?

| | Baseline | Isolating same-sex siblings |
|---|---|---|
| $frac(\alpha_1)$ | -0.234*** | |
| | (0.023) | |
| $frac^2(\alpha_2)$ | 0.209*** | |
| | (0.022) | |
| $fraction(\lambda_1)$ | | -0.535*** |
| | | (0.076) |
| $fraction^2(\lambda_2)$ | | 0.437*** |
| | | (0.075) |
| $girls$ | | -0.096*** |
| | | (0.020) |
| $boys$ | | -0.115*** |
| | | (0.020) |
| $(\alpha_1 + \alpha_2)$ | -0.025*** | |
| $(\lambda_1 + \lambda_2)$ | | -0.098*** |
| Observations | 3105217 | 3105217 |

Dependent variable: duration between births $n$ and $(n+1)$. $frac$: proportion of boys among the previous $n$ children. $girls$: previous $n$ children are all girls. $boys$: previous $n$ children are all boys. $fraction$ is equal to $frac$ iif $frac < 1$. $fraction$ and $fraction^2$ capture the impact of the gender composition among women having at least one child of each gender. Cox estimation, no hazard ratio. Standard errors clustered at the mother level. ***Significant at the 1 percent level. **Significant at the 5 percent level. *Significant at the 10 percent level. Weights. Controls: country, rank of preceding birth, and mother's characteristics: birth cohort, age at preceding birth, religion, family system, union type, education, wealth, area of residence, employment status.

## Appendix D: Full classification

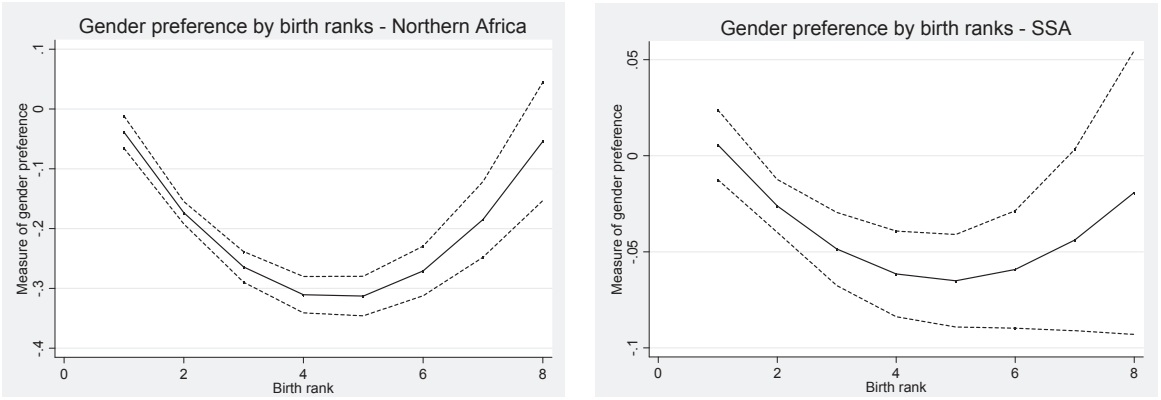
**Table A-3.6:** Which African countries exhibit gender preferences ?

| Country | $\alpha_1 + \alpha_2$ | pvalue of the sum | pvalue joint significance |
|---|---|---|---|
| **$\alpha_1$ and $\alpha_2$ are jointly significant** | | | |
| Egypt | -0.168 | 0.000 | 0.000 |
| Tunisia | -0.099 | 0.013 | 0.000 |
| Burundi | -0.064 | 0.022 | 0.042 |
| Kenya | -0.057 | 0.001 | 0.000 |
| Uganda | -0.055 | 0.001 | 0.001 |
| Mali | -0.050 | 0.005 | 0.000 |
| Mozambique | -0.049 | 0.032 | 0.040 |
| Morocco | -0.048 | 0.017 | 0.000 |
| Tanzania | -0.038 | 0.047 | 0.015 |
| Senegal | -0.038 | 0.018 | 0.045 |
| Zimbabwe | -0.025 | 0.204 | 0.021 |
| Rwanda | -0.022 | 0.228 | 0.000 |
| Guinea | -0.017 | 0.369 | 0.000 |
| Malawi | -0.014 | 0.389 | 0.032 |
| Namibia | -0.002 | 0.934 | 0.097 |
| South Africa | 0.004 | 0.915 | 0.017 |
| Benin | 0.011 | 0.465 | 0.000 |
| Ethiopia | 0.012 | 0.516 | 0.100 |
| Liberia | 0.026 | 0.385 | 0.062 |
| **$\alpha_1$ and $\alpha_2$ are not jointly significant** | | | |
| Swaziland | -0.051 | 0.288 | 0.366 |
| Gabon | -0.019 | 0.576 | 0.754 |
| Zambia | -0.017 | 0.338 | 0.547 |
| Cameroon | -0.015 | 0.422 | 0.157 |
| Nigeria | -0.012 | 0.500 | 0.243 |
| Ghana | -0.009 | 0.652 | 0.673 |
| Madagascar | -0.005 | 0.793 | 0.157 |
| Burkina Faso | -0.002 | 0.923 | 0.798 |
| Niger | 0.002 | 0.887 | 0.569 |
| Congo DRC | 0.005 | 0.894 | 0.259 |
| Congo | 0.010 | 0.738 | 0.278 |
| Sierra Leone | 0.012 | 0.745 | 0.505 |
| Sudan | 0.017 | 0.638 | 0.713 |
| Lesotho | 0.017 | 0.588 | 0.682 |
| Togo | 0.035 | 0.226 | 0.221 |
| Chad | 0.035 | 0.218 | 0.125 |
| Cote d'Ivoire | 0.044 | 0.112 | 0.265 |
| Central African Republic | 0.055 | 0.161 | 0.303 |

Cox estimation. Weights. Controls: country, rank of preceding birth, and mother's characteristics: birth cohort, age at preceding birth, religion, family system, union type, education, wealth, area of residence, employment status. We sort countries by our indicator of gender preferences ($\alpha_1+\alpha_2$). We report the pvalue of the test $(\alpha_1+\alpha_2) = 0$ and the pvalue of the test for joint significance of $\alpha_1$ and $\alpha_2$. The table is split in two between countries exhibiting gender preferences and countries with no preferences.

## Appendix E: Robustness Tests

**Figure A-3.1:** $(\alpha_1 + \alpha_2)$ by birth ranks




The graphs plot an indicator of rank-by-rank gender preferences, using an order 2 polynomial of child's birth rank. The dotted lines represent the 5% confidence intervals. Cox estimation. Standard errors clustered at the mother level. Weights. Controls: country and mother's characteristics: birth cohort, age at preceding birth, religion, family system, union type, education, wealth, area of residence, employment status.

Beware that the scale is not the same in the two graphs.

**Table A-3.7:** Is our indicator driven by the gender of the n$^{th}$ birth in North Africa?

| Sample | Baseline | Decomposition | Decomposition, by rank |
|---|---|---|---|
| $frac_n$ | -0.795*** | | |
| | (0.034) | | |
| $frac_n^2$ | 0.654*** | | |
| | (0.032) | | |
| $frac_{n-1}$ | | -0.467*** | -0.466*** |
| | | (0.038) | (0.039) |
| $frac_{n-1}^2$ | | 0.342*** | 0.340*** |
| | | (0.037) | (0.037) |
| $Boy_n$ | | -0.123*** | -0.106*** |
| | | (0.006) | (0.011) |
| $Boy \times Rank3$ | | | -0.039** |
| | | | (0.017) |
| $Boy \times Rank4$ | | | -0.046** |
| | | | (0.018) |
| $Boy \times Rank5$ | | | -0.026 |
| | | | (0.021) |
| $Boy \times Rank6$ | | | 0.007 |
| | | | (0.024) |
| $Boy \times Rank7$ | | | -0.019 |
| | | | (0.029) |
| $Boy \times Rank8$ | | | 0.024 |
| | | | (0.037) |
| $(\alpha_1 + \alpha_2)$ | -0.141*** | -0.125*** | -0.126*** |
| Observations | 385180 | 286264 | 274358 |

Dependent variable: duration between births $n$ and $(n+1)$. $frac_n$: proportion of boys among the previous $n$ children. $Boy_n$: dummy for birth $n$ is a boy. Sample: North Africa (Morocco, Egypt and Tunisia). In columns 2 and 3, we exclude durations after birth 1 because $frac_{n-1}$ is not defined at that parity. In column 3, we exclude birth orders higher than 8 to have enough observations in each cell by birth order; the birth order of reference is 2. Cox estimation, no hazard ratio. Standard errors clustered at the mother level. ***Significant at the 1 percent level. **Significant at the 5 percent level. *Significant at the 10 percent level. Weights. Controls: country, rank of preceding birth, and mother's characteristics: birth cohort, age at preceding birth, religion, family system, union type, education, wealth, area of residence, employment status.

**Table A-3.8:** Testing the mortality bias

| | Baseline | Test |
|---|---|---|
| $frac(\alpha_1)$ | -0.234*** | -0.321*** |
| | (0.023) | (0.025) |
| $frac^2(\alpha_2)$ | 0.209*** | 0.293*** |
| | (0.022) | (0.024) |
| dead child | | 0.009 |
| | | (0.016) |
| $frac \times$ dead child | | 0.257*** |
| | | (0.059) |
| $frac^2 \times$ dead child | | -0.236*** |
| | | (0.054) |
| $(\alpha_1 + \alpha_2)$ | -0.025*** | -0.028*** |
| Observations | 3105217 | 3105217 |

Dependent variable: duration between births $n$ and $(n+1)$. $frac$: proportion of boys among the previous $n$ children. dead child: dummy for at least one dead child among the previous $n$ children. Cox estimation, no hazard ratio. Standard errors clustered at the mother level. ***Significant at the 1 percent level. **Significant at the 5 percent level. *Significant at the 10 percent level. Weights. Controls: country, rank of preceding birth, and mother's characteristics: birth cohort, age at preceding birth, religion, family system, union type, education, wealth, area of residence, employment status.

**Table A-3.9:** Testing the mortality bias – by groups of countries

| | Preference for variety | | Son preference | |
|---|---|---|---|---|
| | Baseline | Test | Baseline | Test |
| $frac(\alpha_1)$ | -0.200*** | -0.166*** | -0.418*** | -0.591*** |
| | (0.050) | (0.055) | (0.023) | (0.025) |
| $frac^2(\alpha_2)$ | 0.203*** | 0.163*** | 0.340*** | 0.512*** |
| | (0.048) | (0.053) | (0.022) | (0.024) |
| dead child | | 0.025 | | -0.035** |
| | | (0.034) | | (0.016) |
| $frac \times$ dead child | | -0.174 | | 0.704*** |
| | | (0.126) | | (0.062) |
| $frac^2 \times$ dead child | | 0.210* | | -0.693*** |
| | | (0.111) | | (0.058) |
| $(\alpha_1 + \alpha_2)$ | 0.003 | -0.003 | -0.078*** | -0.079 *** |
| Observations | 773577 | 773577 | 1167669 | 1167669 |

Dependent variable: duration between births $n$ and $(n+1)$. $frac$: proportion of boys among the previous $n$ children. dead child: dummy for at least one dead child among the previous $n$ children. Cox estimation, no hazard ratio. Standard errors clustered at the mother level. ***Significant at the 1 percent level. **Significant at the 5 percent level. *Significant at the 10 percent level. Weights. Controls: country, rank of preceding birth, and mother's characteristics: birth cohort, age at preceding birth, religion, family system, union type, education, wealth, area of residence, employment status. Countries in group "Preference for variety": Zimbabwe, Rwanda, Guinea, Malawi, Namibia, South Africa, Benin, Ethiopia and Liberia. Countries in group "Son preference": Egypt, Tunisia, Burundi, Kenya, Uganda, Mali, Mozambique, Morocco, Tanzania and Senegal.

## Appendix F: In which countries does the gender composition of the first two children predict subsequent fertility choices?

One extension of our analysis is to consider the gender composition of previous children as an instrument for future fertility choices in order to estimate the impact of fertility on another outcome. In which African countries are gender preferences strong enough for the first stage to hold? We examine the situation after the second birth. Since $Frac_n$ is not at all continuous when $n = 2$, we estimate an alternative proportional hazard model:

$$\lambda(t) = \lambda_0(t) \times \exp(\beta_{girls}.Girls + \beta_{boys}.Boys + \theta.X_2)$$

Where $Girls$ is a dummy equal to 1 if the first two children are girls, $Boys$ is a dummy equal to 1 if the first two children are boys. We do not reweigh the observations here, because the unit of observation is the mother. Robust standard errors are clustered at the finest geographical level defined in DHS (DHS cluster). Women having exactly 1 boy and 1 girl are the reference category. If $\beta_{girls} > 0$ and $\beta_{boys} > 0$, there is evidence of a taste for balance, because the lowest hazard rate – hence the longest expected interval before the third birth – is reached by women having children of both sex. Then, from the relative values of $\beta_{boys}$ and $\beta_{girls}$, we can infer the existence of son or daughter preference.

Not surprisingly, the gender composition of the first two children is a strong predictor of next birth spacing in North Africa: Morocco, Tunisia and Egypt systematically display a strong son preference. Couples in Mali, Nigeria and Zimbabwe also wait significantly less before the third birth when they have at first two daughters vs. two sons. Then, the instrument would work in Benin, Guinea, Rwanda, Malawi and South Africa: they exhibit preferences for variety. Interestingly, some countries display daughter preference after the second birth: Ethiopia, Namibia, and Cote d'Ivoire. But in the vast majority of countries, the gender composition of the first two children does not influence the duration before third birth.

How to explain the discrepancy between the classification mentioned above and the one illustrated in Figure 3.3? When we use only intervals after the second birth, we lose some power to detect small magnitudes, as compared to the specification exploiting all parities. Mechanically, there are more countries in which we find no evidence of gender preferences at rank 2. Furthermore, as shown in section 3.6.3, the impact of gender preferences is weaker at rank 2 than at higher ranks. However, two specific cases are worth mentioning: in Nigeria and Cote d'Ivoire, gender preferences (respectively, son preference and daughter preference) are significant at rank 2 but disappear at higher ranks.

# CONCLUSION

This PhD thesis provides new evidence on health progress in Africa in the 20th century. It focuses on two potential challenges when looking at the impact of health policies: *(i)* the endogeneity of health policies under colonial rule and *(ii)* the selective mortality bias. It also *(iii)* studies gender preference across time and space in the African continent.

Regarding the endogeneity of health policies, the first chapter studies the main determinants of health investments between 1904 and 1958 in former French West Africa. It demonstrates that there exists a very general strategy regarding the provision of colonial services. Colonial health policies implemented in former French West Africa were far from being exogenous. A detailed analysis of the colonial health system shows that the development of health policies took place in close relation with the broader development of all colonial policies. The common factors to all investments have to do with the colonial administration's preference for path dependence, investments' returns to scale, the diseases' contagion risk and the demand for colonial services. This paper also suggests that colonial investments' decisions were guided by a general – rather than specific – principle of investments' complementarity; and that there was no specialization of districts in one type of investments. The allocation of health inputs is specific in only two dimensions. First, medical staff is used as a means of colonial "coverage". Second, all health investments follow a "diversification" strategy related to the long-lasting effect of prevention, which does not exist for education.

To tackle the second question of the thesis, I exploit West African DHS surveys to study the relationship between adult height and under-five mortality, in Chapter 2. I am able to bring forward a positive height-mortality correlation in the 1980's West Africa. This first intuition of selective mortality is confirmed by the estimation of an original model of height differential between survivors and deceased. Results imply that selective mortality could be large enough to mask significative height increases in the 1980's West Africa. Hence, in high-mortality settings, the selective mortality bias can be such that survivors' height trends are misleading. The general implication of this result is that any study finding that health interventions have a null or

negative impact on long-term outcomes should discuss the selective mortality bias with great caution.

Finally, the last question of this thesis is tackled in Chapter 3, co-authored with Pauline Rossi. We develop a new indicator of gender preferences based on birth spacing. From this indicator, we analyse the heterogeneity of preferences across time and space. This research provides evidence that son preference is strong and increasing in North Africa. Sub-Saharan African countries display a preference for variety or no preference at all. Regarding socioeconomic factors, wealthier and more educated women display the same type of preferences as the others, but the magnitude of their preferences is much larger. Then, in sub-Saharan Africa, there is no correlation with religion: Muslims exhibit the same preferences as other religious groups. On the other hand, traditional kinship structure accurately predicts the nature of preferences: son preference prevails in patrilineal ethnic groups only.

In parallel to these research questions, I have worked on a further project on the long-term evolution of African living standards under the colonial rule. The main conclusions of this dissertation also led to new research questions, to which future projects will try to answer. The reader will find below the detail of these on-going and future projects.

Over the last few years, I have worked on a project untitled "African Living Standards under the French Empire: Evidence from Recruits to the Tirailleurs Sénégalais", which is co-authored with Denis Cogneau and Alexander Moradi. For this project, we use conscripts data from the Colonial French Army on former French West Africa. Military archives are the only source that gives a large coverage of space and time regarding living standards in West Africa before colonial independence. For the eight colonies of former French West Africa, they contain one million individual conscripts files for the cohorts born in 1880-1940. These data allow to study the evolution of nutrition and living standards in the region, through anthropometric studies.

Before analyzing long-term height trends, we need to ask what is the representativeness of these data. There is a specific sample-selection bias for military archives due to the selection of physically fit men into army. In former French West Africa, after WW1, formal conscription was progressively put in place during the years 1920 to 1925. A recruitment target was fixed for the whole region by the Ministry of Defense in Paris; this global target was then distributed across the eight colonies by the general governor, and in a third step, distributed across the colonial districts by each colony governor. Within each colonial district, the formal procedure then involved three other stages. First, the district administrator organized the enumeration of

the eligible 20 year-old male population. Second, mobile draft commissions were organizing medical exams checking for fitness. Third, once volunteers had applied, a lottery rate was fixed according to both the number of fit and the target. The fit who had drawn the wrong numbers were drafted for three years ("first portion"), while the remaining others were considered as reservists ("second portion"). Aside to individual soldier files, we also collected data on these drafting commissions for the years 1920 to 1948, kept at the Archives of Senegal in Dakar. These commissions had recruitment targets in mind, so that they could be rather selective in terms of physical requirements, provided that the number of fit would remain well above these targets.

Selection into conscription can be problematic for anthropometric studies if the required level of fitness changes over time. In order to address this issue, we compute inverse Mills ratios for the probability of being drafted (as fit), and for the more general probability that individual height is measured. These ratios are obtained from colony-specific probit regressions, at the district level, for years 1895 to 1960. Then, we add these inverse Mills ratios in our height equations, in order to correct for selection. Preliminary results imply that high mean heights observed for birth cohorts before 1905 or after 1930 (conscripted before 1925 or after 1950) are due to selection or small sample sizes. Moreover, for birth cohorts 1905 to 1930, height stagnation dominates. Cote d'Ivoire and Burkina Faso are possible exceptions, but the selection correction makes their trends non-significant.

Another development of this research is the analysis of the long-term implications of colonial health policies in former French West Africa. Using DHS (Demographic and Health Surveys) data to cover the post-colonial period, as well as colonial health reports and conscripts files for the colonial period, I am able to build a pseudo-panel of West-African districts from 1905 until now.

This pseudo-panel contains health inputs variables: *(i)* vaccinations, medical staff and health facilities during colonial times, from colonial reports; *(ii)* medical staff and smallpox vaccinations during the 1970's, from WHO reports; *(iii)* vaccination rates, household sanitation and bednet use from the 1980's, using DHS data; and *(iv)* availability and proximity of health facilities and staff from the 1980's, using DHS data for some country$\times$year providing specific questionnaires on this matter. This panel also contains outcomes variables: *(i)* disease cases and deaths during colonial times, from colonial reports; *(ii)* average male height at the district level for cohorts 1880-1940, from conscripts files; *(iii)* smallpox casualties during the 1970's, from WHO reports; *(iv)* child height, for cohorts born from the 1980's, using DHS data; and *(v)* female adult height, for cohorts born from the 1940's, using DHS data.

These data can then be used to identify the short and long-term impact of colonial health policies, while looking at health from a multi-dimensional perspective. In continuity with Chapter 1, this work will allow me to look into within-colonization and post-independence history: what has happened from early colonial investments to today's outcomes? The pseudo-panel of health inputs and outcomes will cover both colonial and post-colonial period, with several points in time within each of these periods. These features will allow me to look into whether spatial differences persisted over time, or whether homogenization took place. I then intend to look into the multi-dimensional consequence of health on long-term health and on education and fertility.

Last, three research projects build on the work started in this dissertation with Pauline Rossi. Namely, we want to discuss several aspects of gender preferences that were revealed in our publication.

First, our results show that the reinforcement of son preference in Egypt coincides with a reduction in family size. We would like to investigate if the relationship is causal, as suggested by Jayachandran (2014). We could look at other indicators of son preference, such as sex ratio, declared preferences, differential survival rates and health outcomes between boys and girls; and study how they relate to the fall in fertility rates.

Second, we are willing to study more precisely the incidence of daughter preference in Africa. Our work reveals that matrimonial and matrilocal societies are characterized by daughter preference. Moreover, in some specifications, Cote d'Ivoire was not far from revealing daughter preference. Further work is needed to document whether daughter preference can be exhibited in some specific regions, looking at a more disaggregated level than the country.

A third project will study the link between conflicts and gender preferences in Africa. Over the last decades, the African continent has suffered from the occurrence of many internal conflicts, leading to a large number of casualties. Theoretically, such conflicts can affect the anticipated risk of children's mortality for couples at a reproductive age. Hence, conflicts can have an impact on fertility preferences. There might be an impact on the quantity of children, as well as on gender preferences, given that boys and girls are not affected by conflicts in the same way. In particular, several of these conflicts involve child soldiers. For the purpose of this work, we will be able to match DHS data on gender preferences to conflict data on Africa. In particular, we plan to use the Cross-National Time-Series data (CNTS), as well as the Armed Conflict Location and Event data (ACLED) and the UCDP/PRIO Armed Conflict dataset.

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